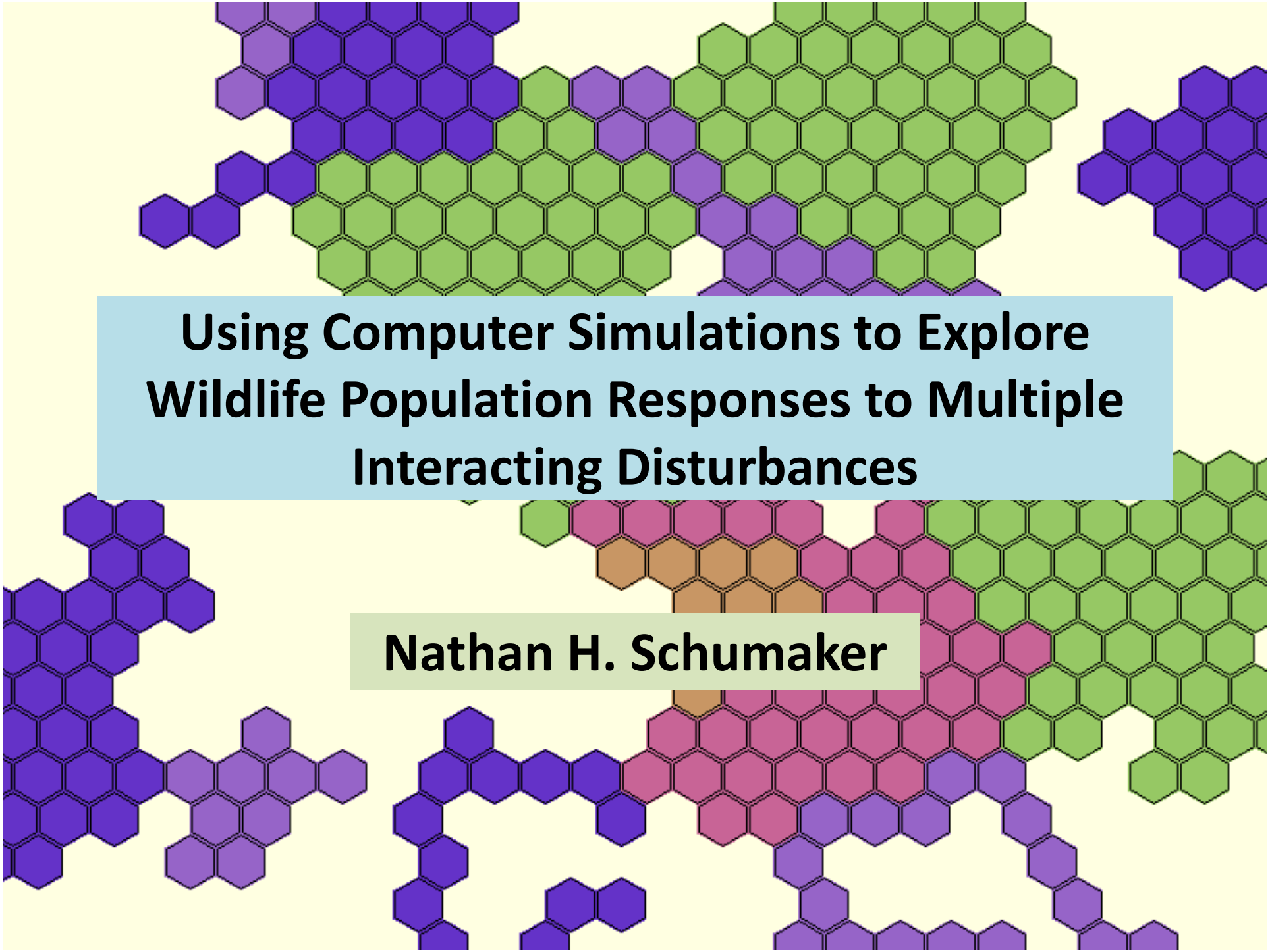


# Using Computer Simulations to Explore Wildlife Population Responses to Multiple Interacting Disturbances

Nathan H. Schumaker

# The Problem

**Conservation biology involves forecasting, and forecasting necessitates the use of models.**

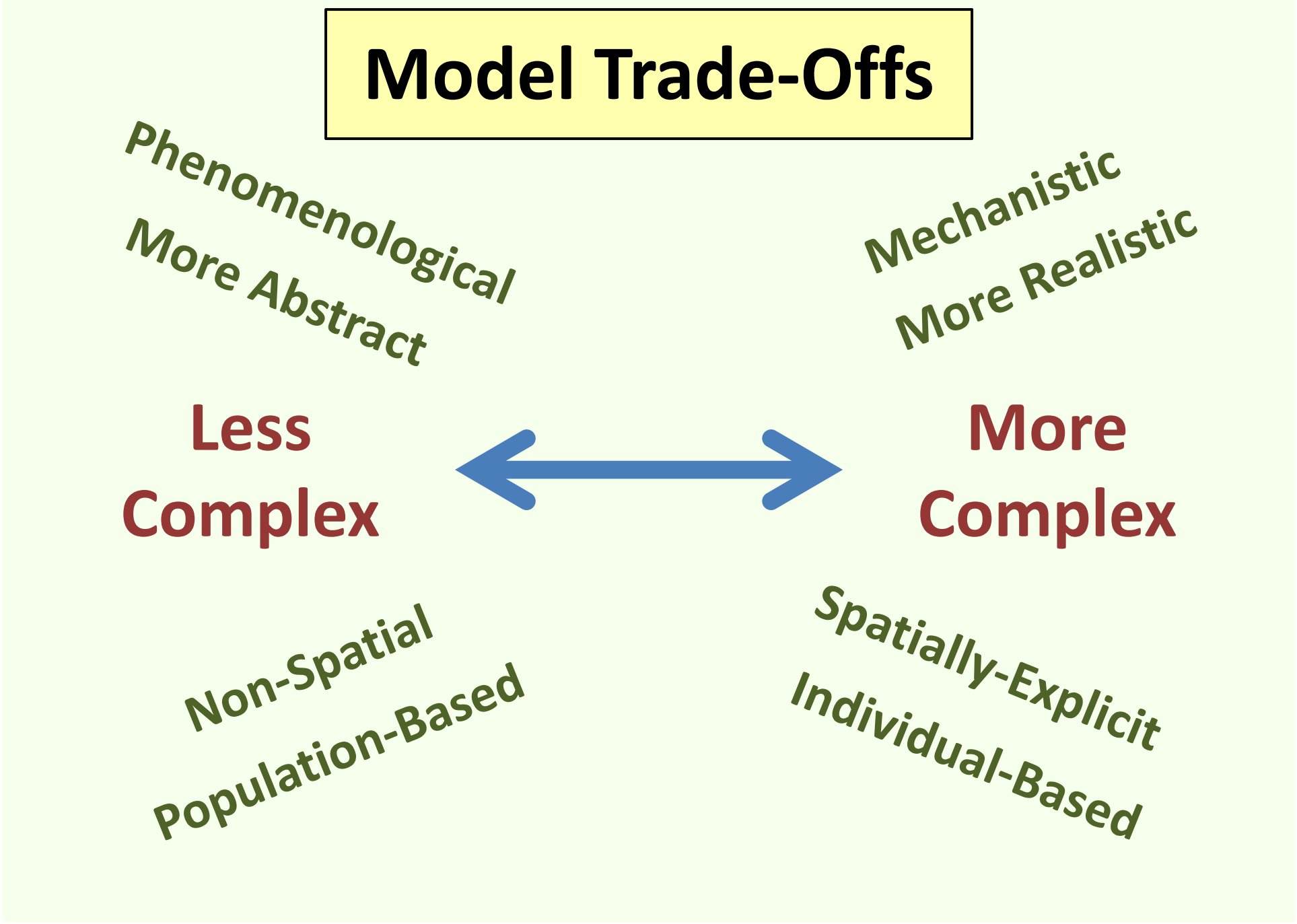
**The use of models (for PVAs, recovery plans, etc.) introduces a number of complications…**

- **Model selection**
- **Model sophistication**
- **Realism vs. complexity**
- **Defensibility**
- **Usability**

# The Need to Address Real-World Problems Drives the Development of New Models

- **Threatened Species.** Recovery planning must balance realism with defensibility.
- **Zoonotic Disease.** Forecasting spread rates and impacts requires sophisticated modeling tools.
- **Off-Target Pesticide Impacts.** Vary based on complex interactions with other stressors.
- **Conservation Genetics.** Future models must be both spatially-explicit and individual-based.

# Model Trade-Offs



# Complications
# (that I deal with…)

- **Landscapes.** They are dynamic; structure matters; features change with life history
- **Populations.** They have complex, diverse life histories, and can interact
- **Disturbances.** Can vary in space and time; there can be lots of them; they often interact
- **Methodology.** Must be defensible and usable, plus have value to decision-makers, etc.

# Some Classes of Models

**I'm going to draw examples from the application of a specific *spatially-explicit* and *individual-based* modeling tool. But there are lots of alternative model structures…**

- **Non-spatial models**
- **Spatially-implicit models**
- **Population models (not individual-based)**

# Non-Spatial Models

## Advantages

- Lots of analytic tools
- Can get a solution
- Easy to communicate

$$N(t+1) = MN(t)$$

$$N = \begin{bmatrix} x \\ y \\ z \end{bmatrix} \quad ; \quad M = \begin{bmatrix} m_{1,1} & m_{1,2} & m_{1,3} \\ m_{2,1} & m_{2,2} & m_{2,3} \\ m_{3,1} & m_{3,2} & m_{3,3} \end{bmatrix}$$

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{k}\right)$$

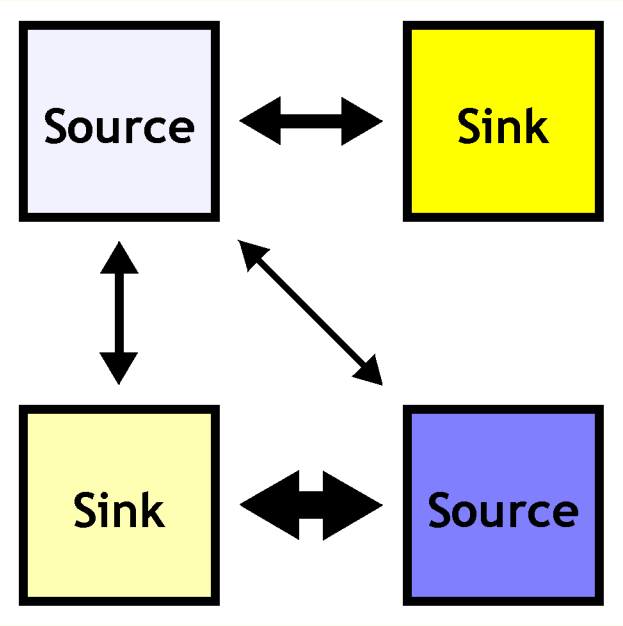
## Disadvantages

- Constrain realism
- Solutions can be hard to obtain

# Spatially-Implicit Models

**Examples**

- Patch occupancy models
- Cellular automata
- Metapopulation models




**Advantages**

- Significantly simpler than fully spatial models

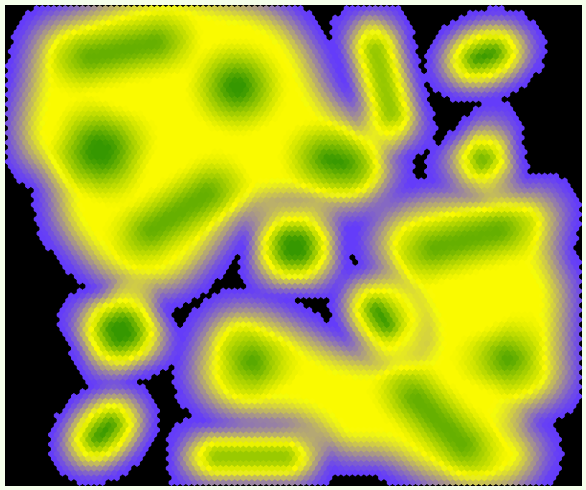
**Disadvantages**

- Limits the influence that landscape structure can have on the model dynamics

# Spatially-Explicit Models

**Examples**

- GIS-based models
- NetLogo
- HexSim



**Advantages**

- Biological / ecological realism

**Disadvantages**

- Additional complexity

# Population Models

**Examples**

$$\frac{\partial N}{\partial t} = D\frac{\partial^2 N(x,t)}{\partial x^2} + rN\left(1 - \frac{N}{k}\right)$$

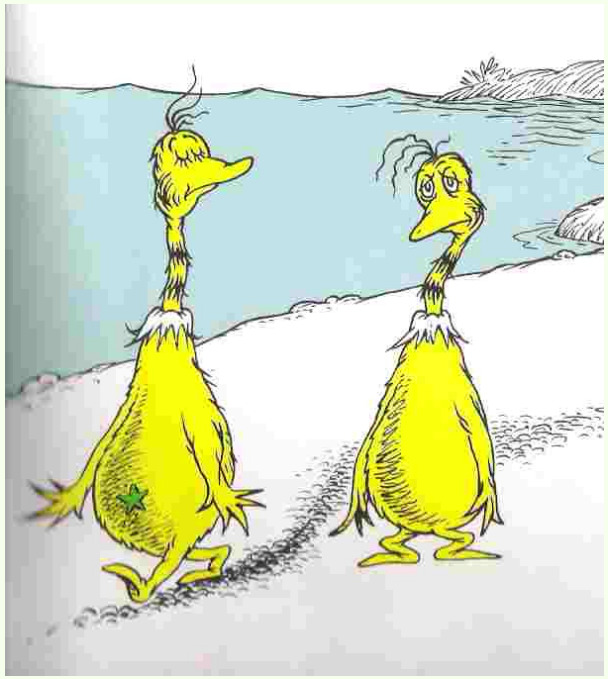
**Advantages**

- **Models are well-defined and parameter space is finite**

**Disadvantages**

- **Models become increasingly complex and hard to understand as realism is added (even when added in limited quantities)**

# Individual-Based Models



**Examples**

- Vortex
- CDPop
- HexSim

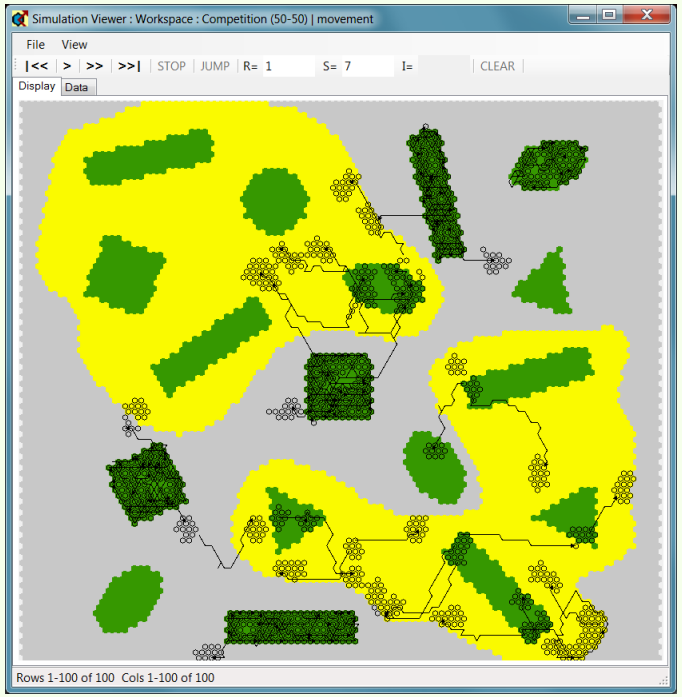
**Advantages**

- Much more mechanistic

**Disadvantages**

- More work to construct and parameterize

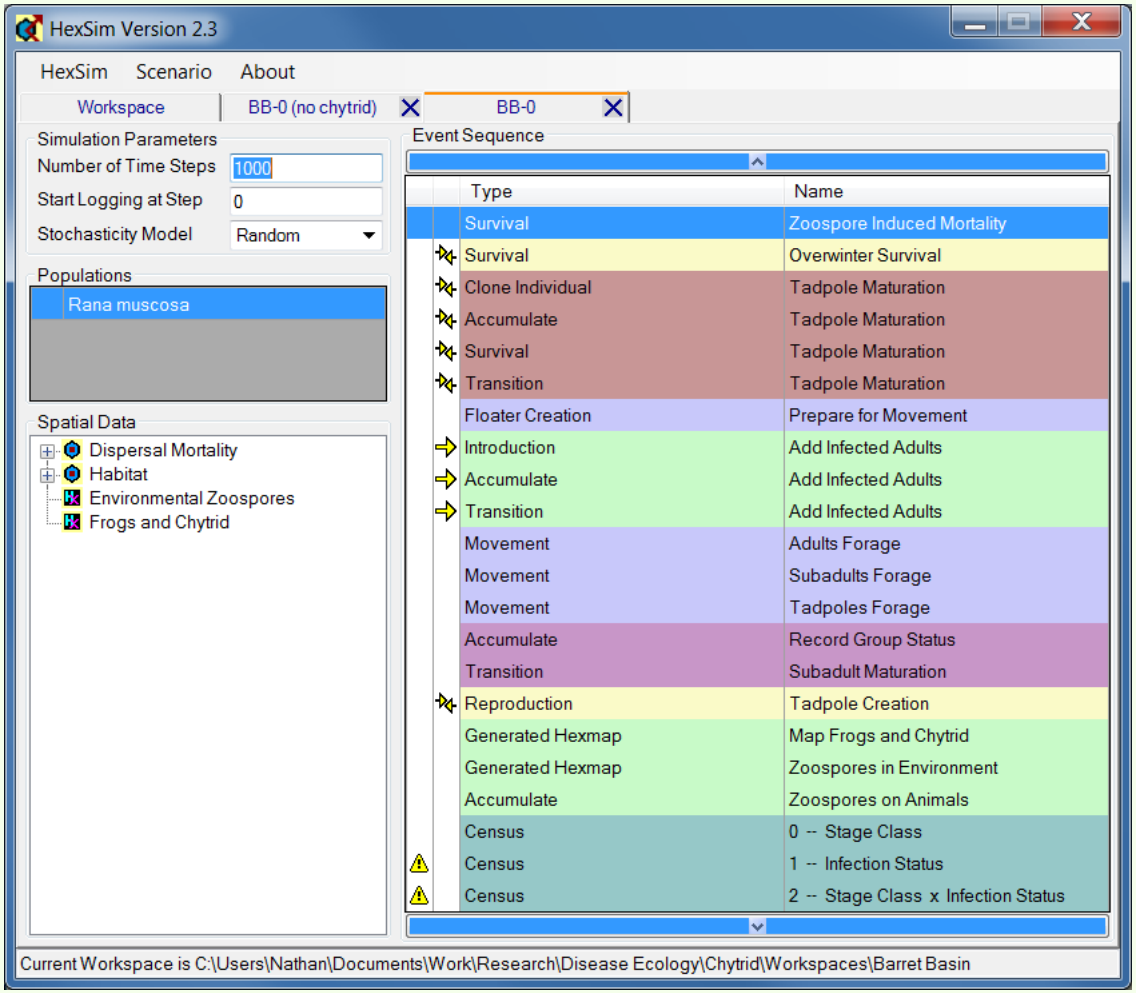
# Spatially-Explicit *and* Individual-Based Models




- Many have been developed, but few are carefully designed, flexible, and reusable
- This class of models make it possible to simulate impacts on a population of multiple interacting stressors
- This class of models is necessary for developing realistic simulations of disease spread, landscape genetics, and other dynamic spatially processes

# HexSim Model

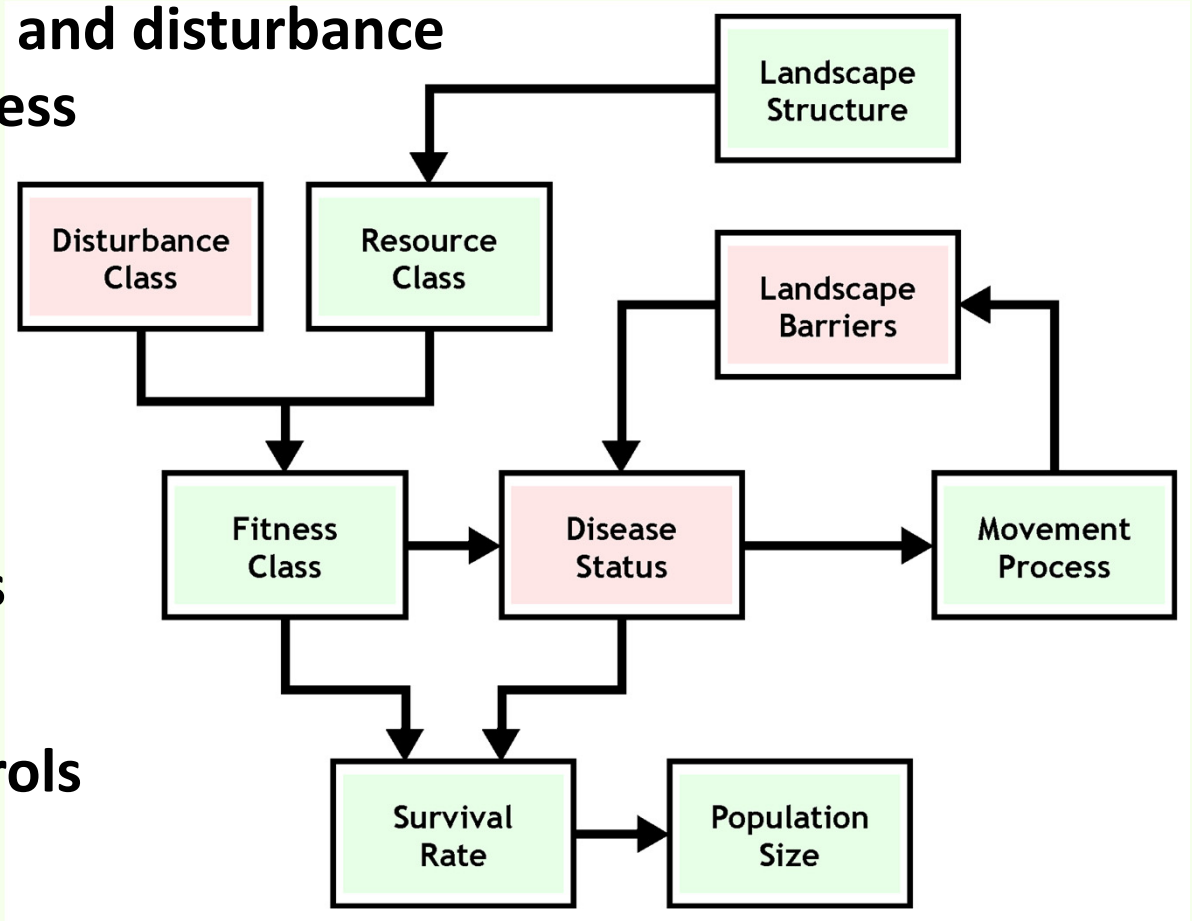
- Modeling framework
- Range of applications
- Trait-based design
- Multiple interacting stressors
- Modern interface



# HexSim Mechanics

## -- A Hypothetical Example --

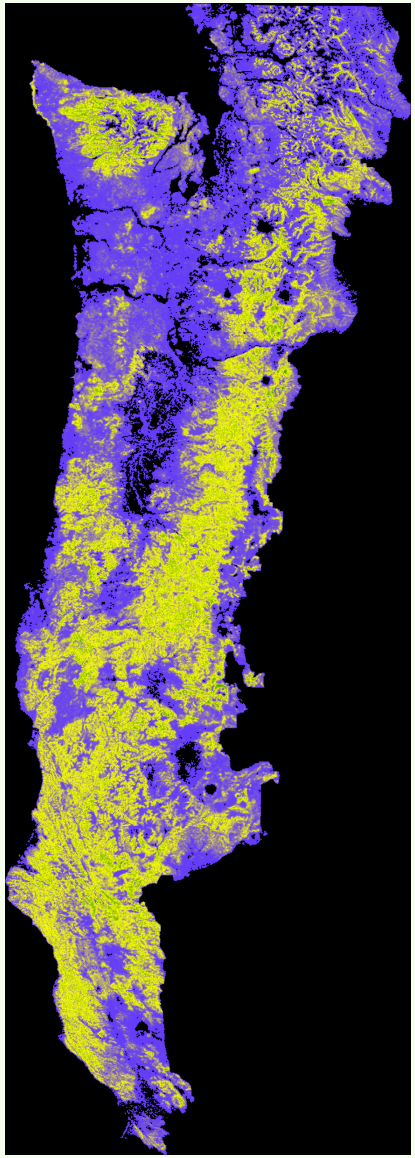
- Landscape structure and population size together limit resource acquisition
- Resource acquisition and disturbance together control fitness
- Fitness and disease control vital rates
- Roads impact the movement process
- Movement mediates disease spread
- Disease spread controls population size

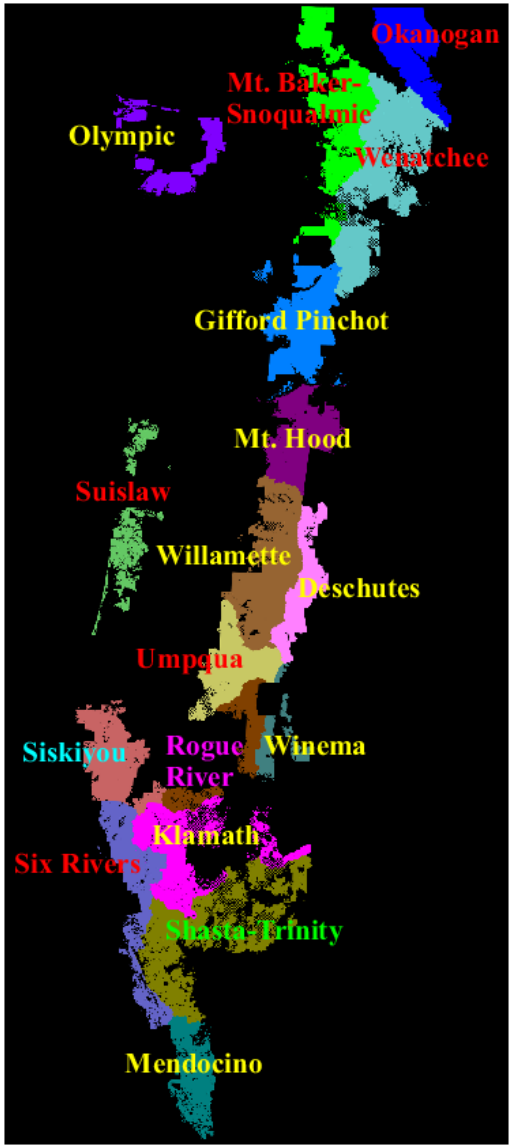


# Examples I'll Discuss Today

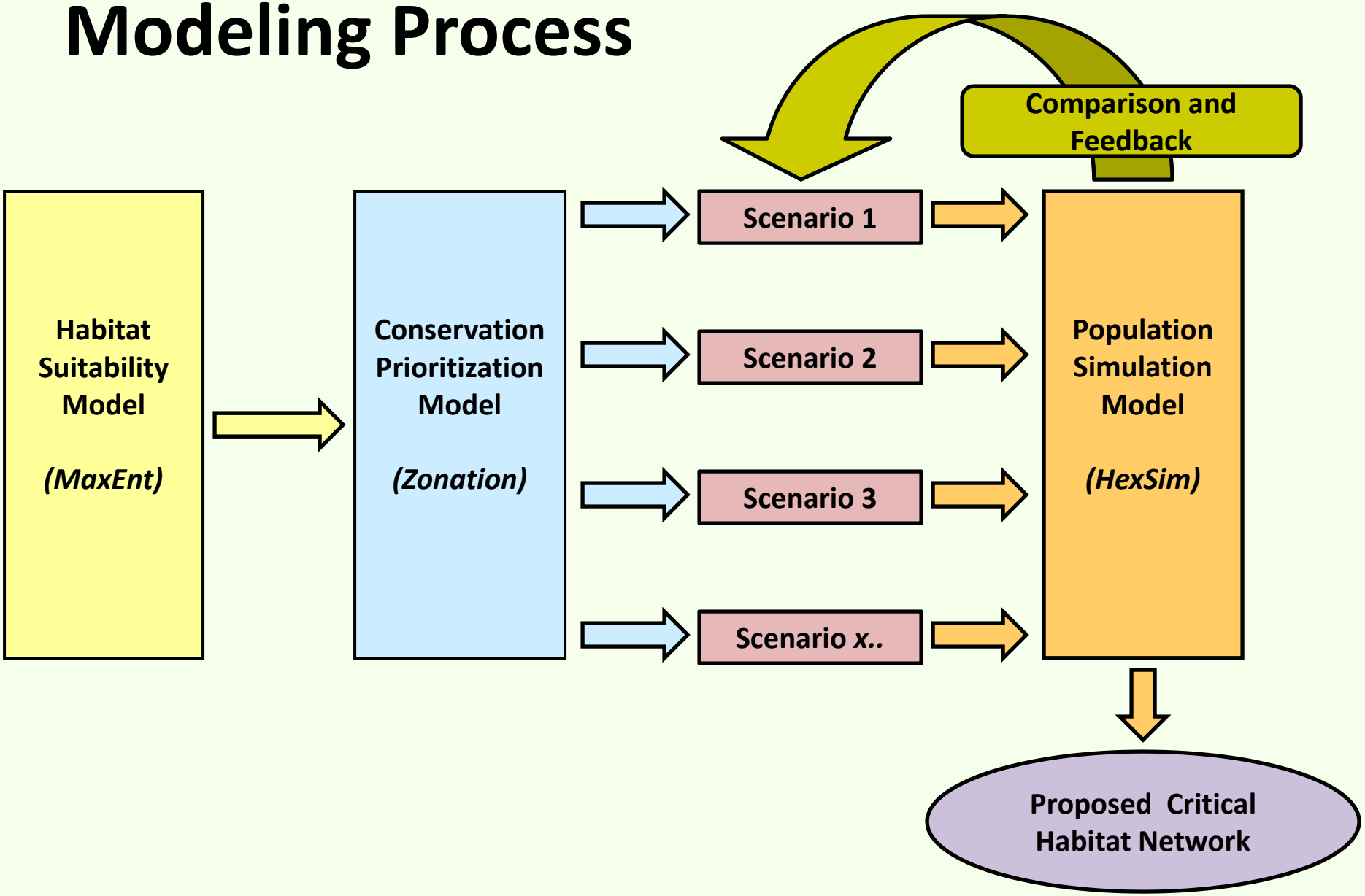
- **Threatened Species.** **HexSim was used in the recovery planning and critical habitat designation for the Northern Spotted Owl.**
- **Wildlife Disease.** **Frog / chytrid model is in development. Other models are on the way.**
- **Conservation Genetics.** **HexSim will make more sophisticated studies possible.**

# Northern Spotted Owls

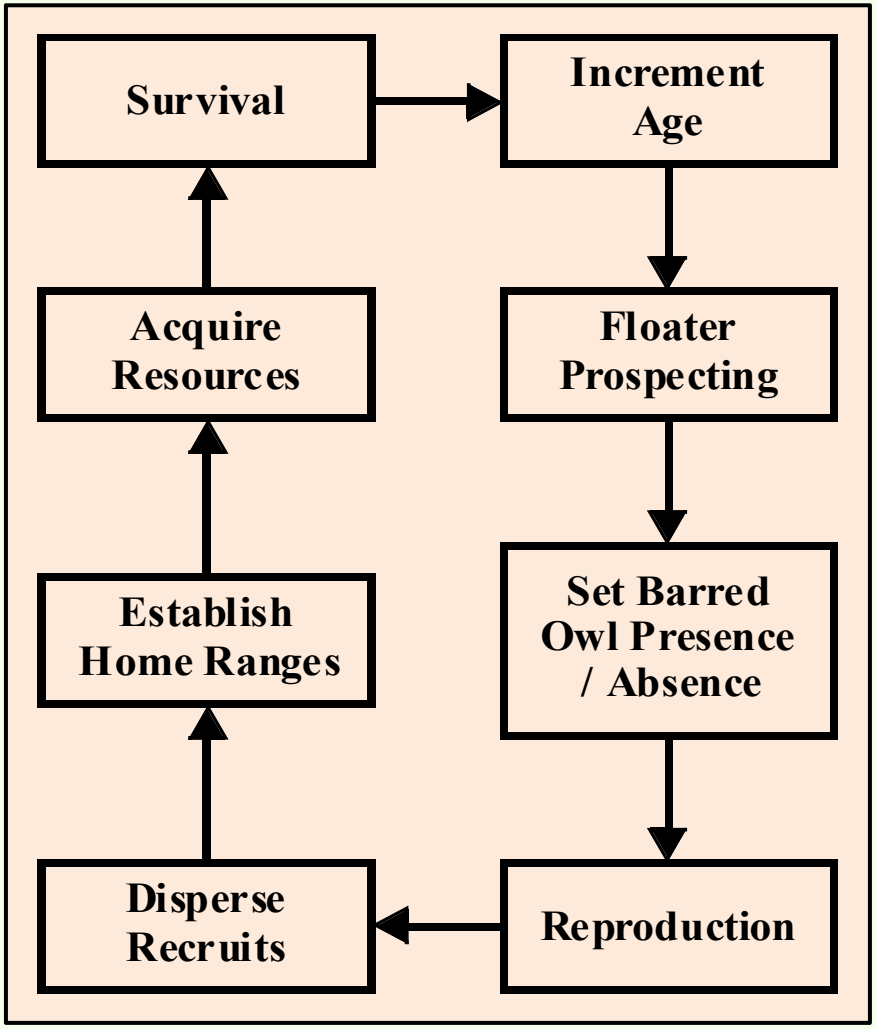


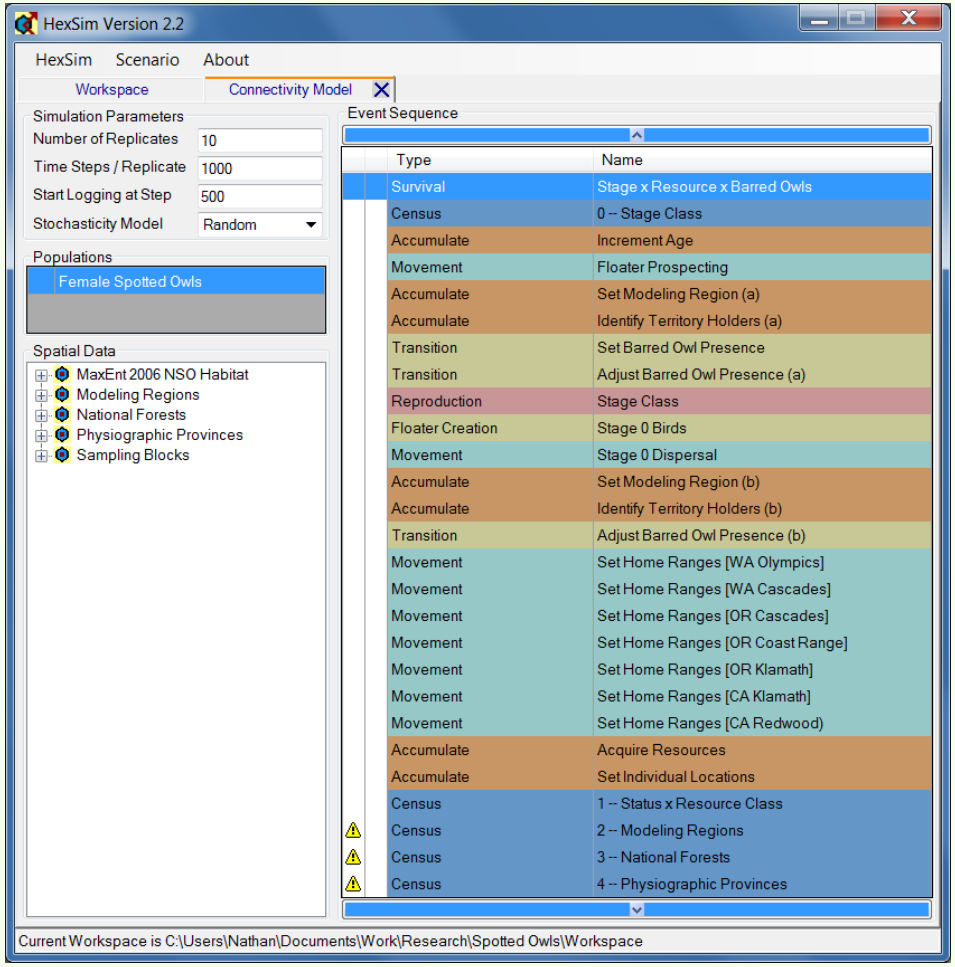


# The Planning & Modeling Process

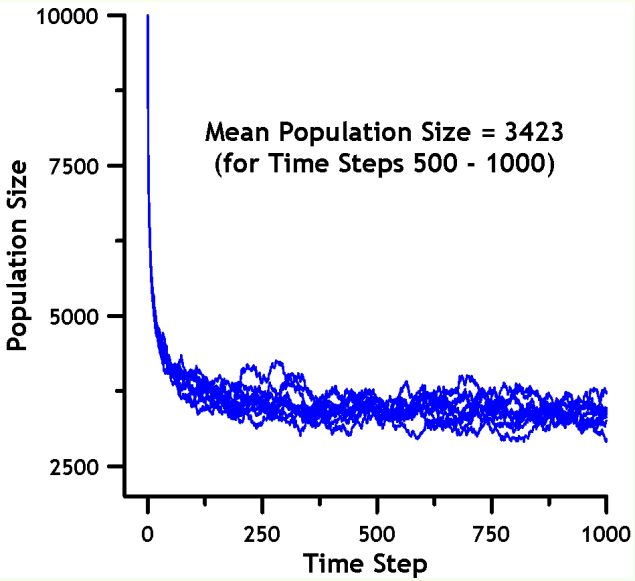


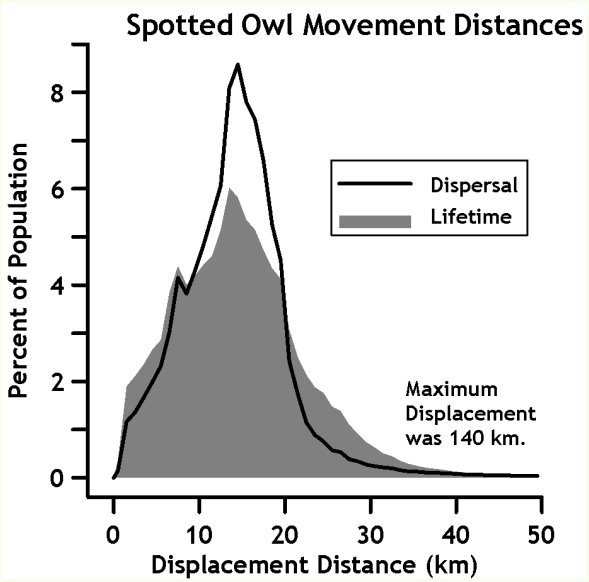
# HexSim Spotted Owl Model

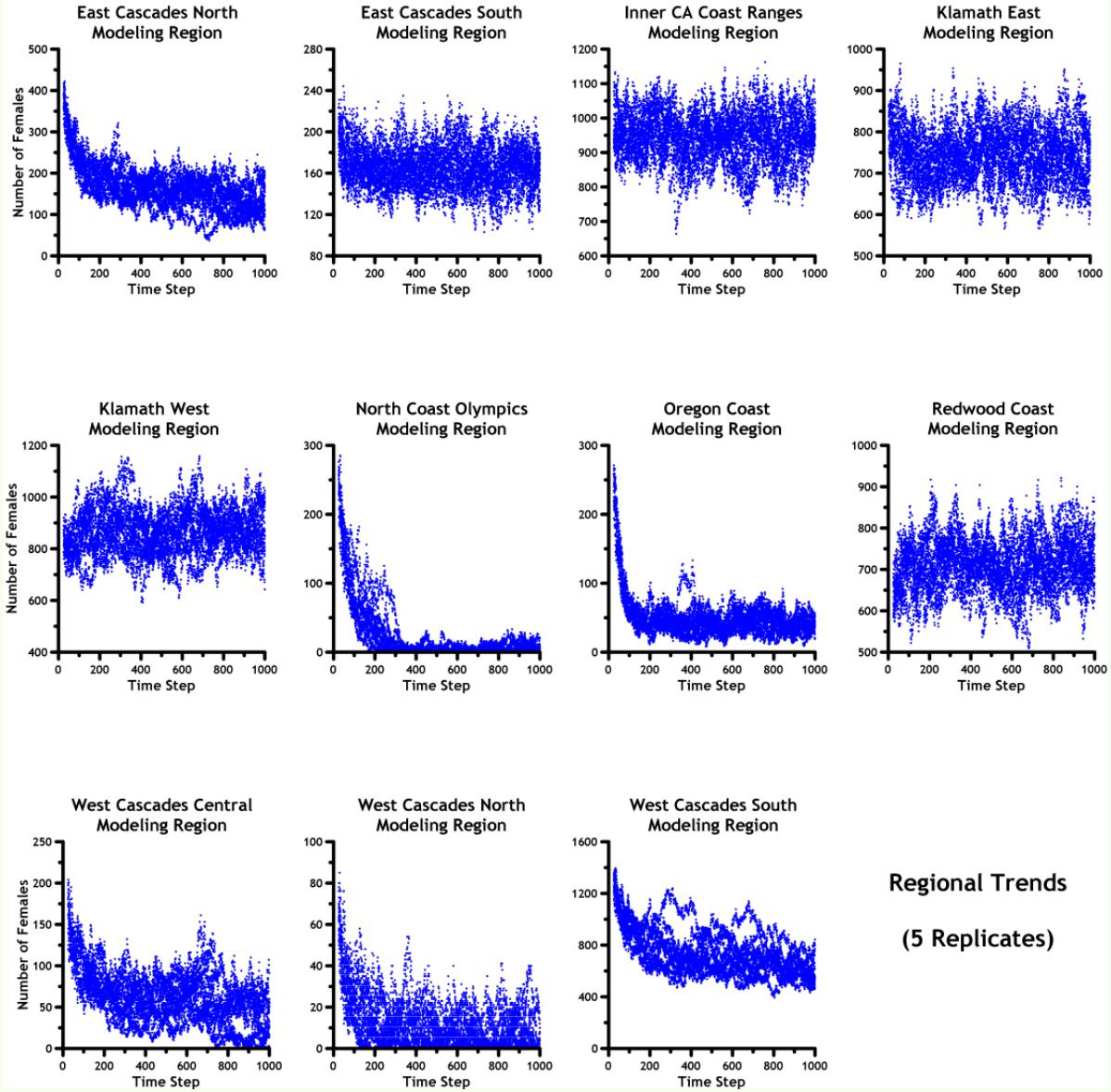


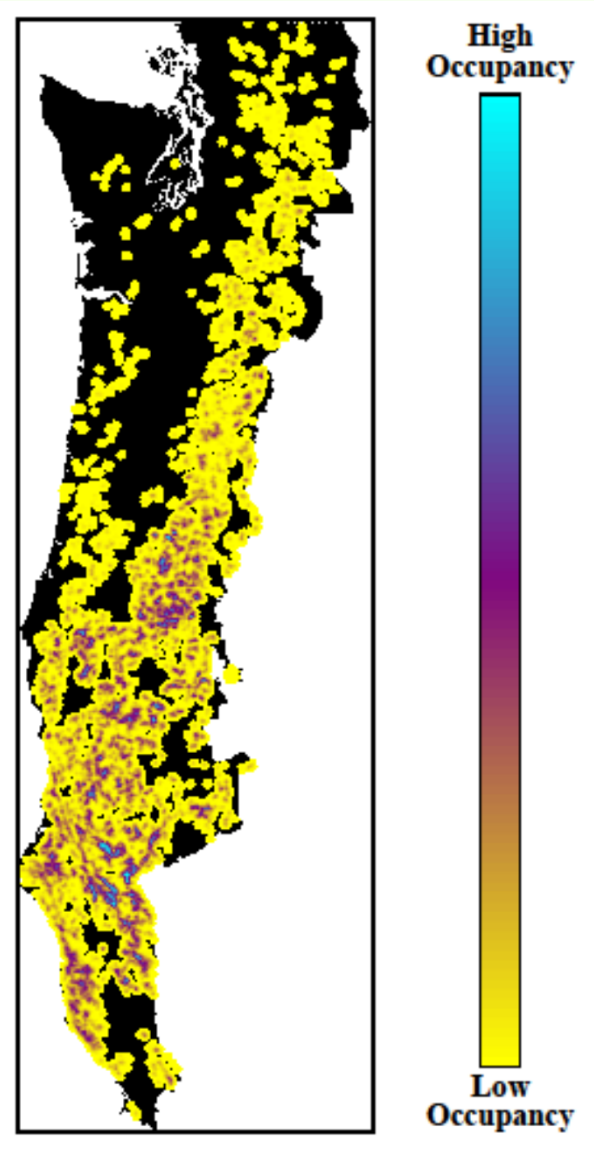
# Model Verification

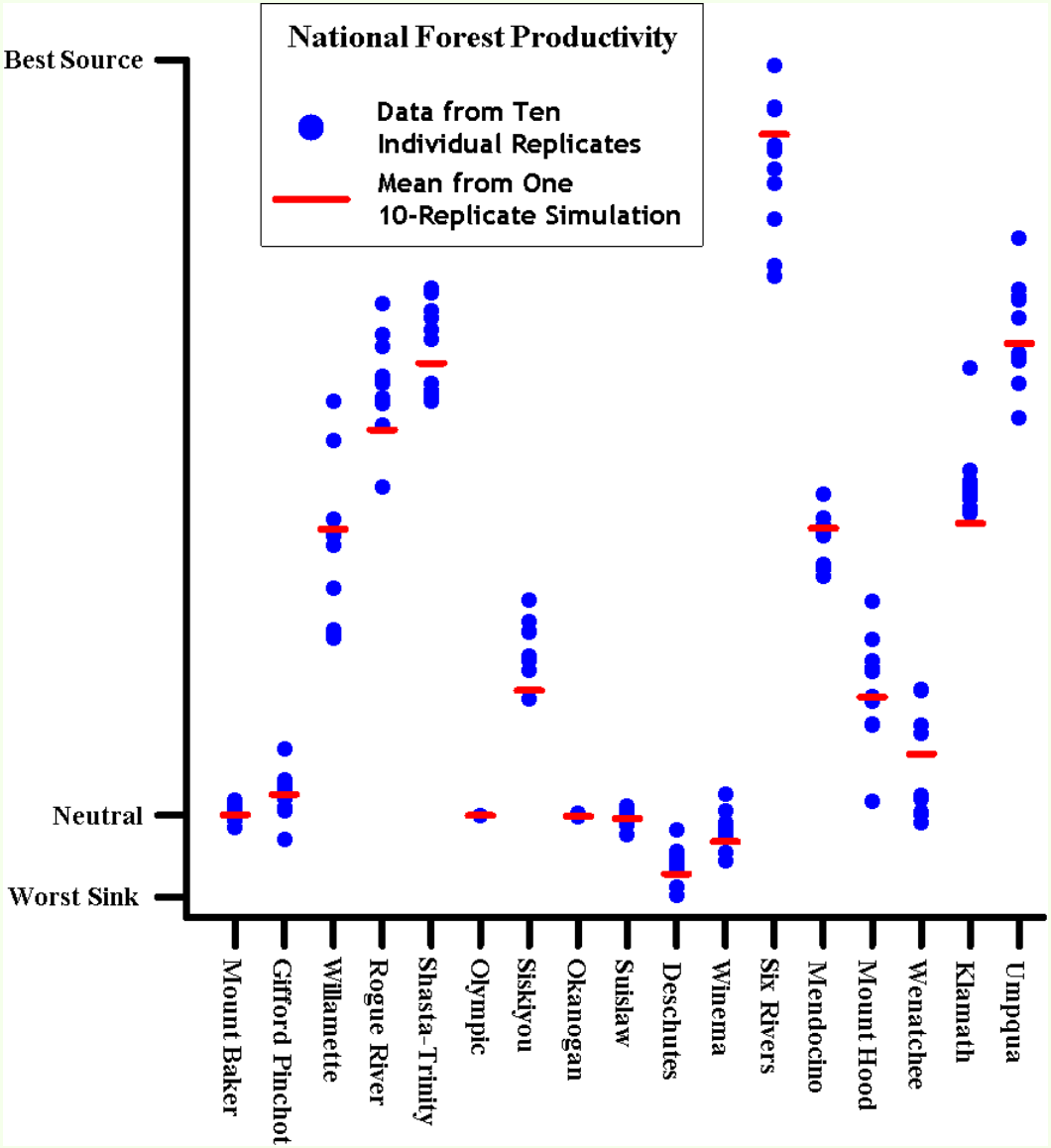


# Source-Sink Analysis
# (National Forests)



# Demographic Sinks

Most Severe Sink

Most Benign Sink

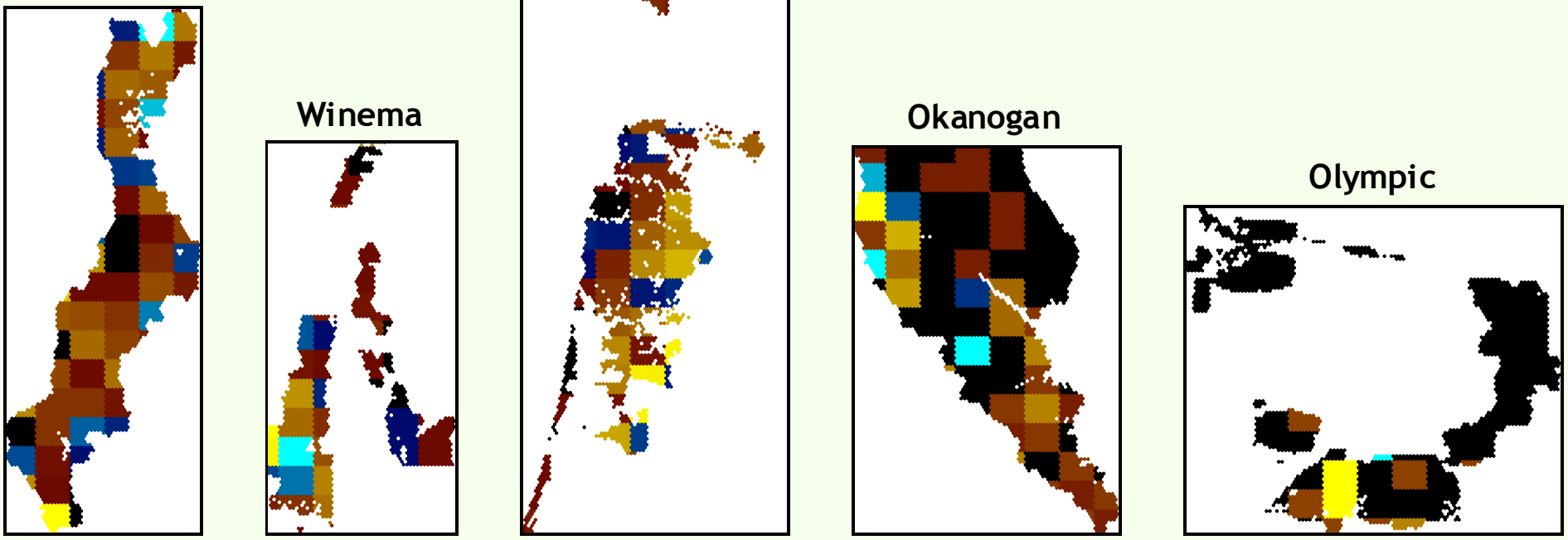
Deschutes

Winema

Siuslaw

Okanogan

Olympic



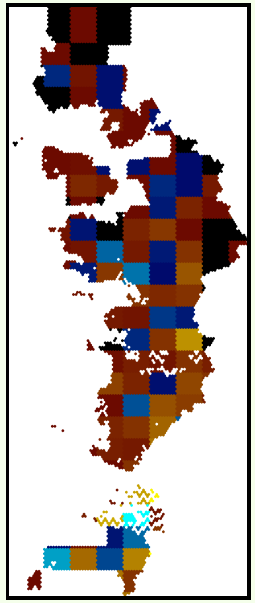
Productivity (10 replicates)

Worst Sink — Neutral — Best Source

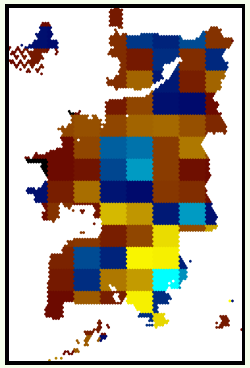
Weakest Source

# Demographic Sources

Mount Baker-Snoqualmie

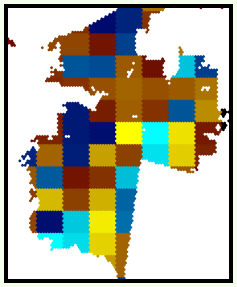


Gifford Pinchot

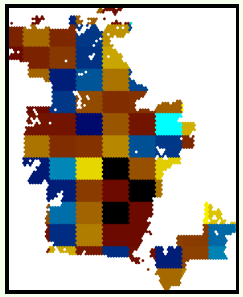


Wenatchee

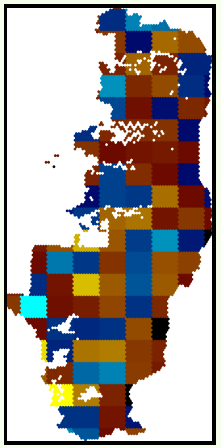
Mount Hood



Siskiyou

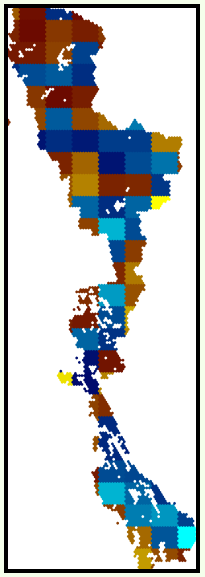


Willamette

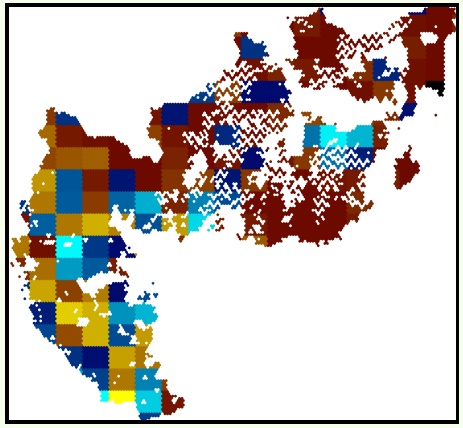


Mendocino

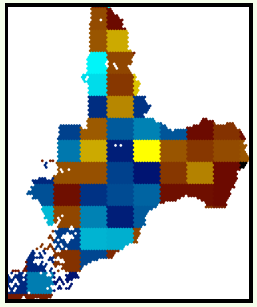
Six Rivers



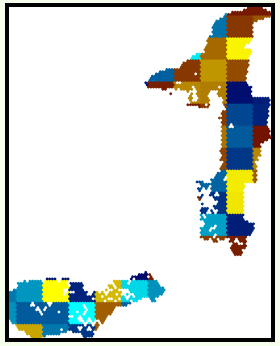
Shasta-Trinity



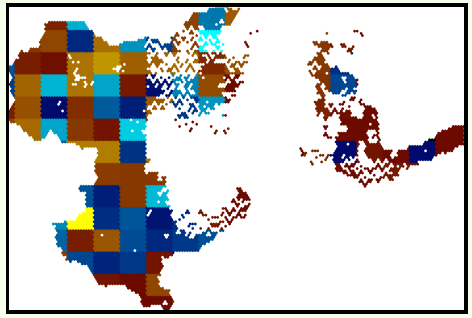
Umpqua



Rogue River



Klamath



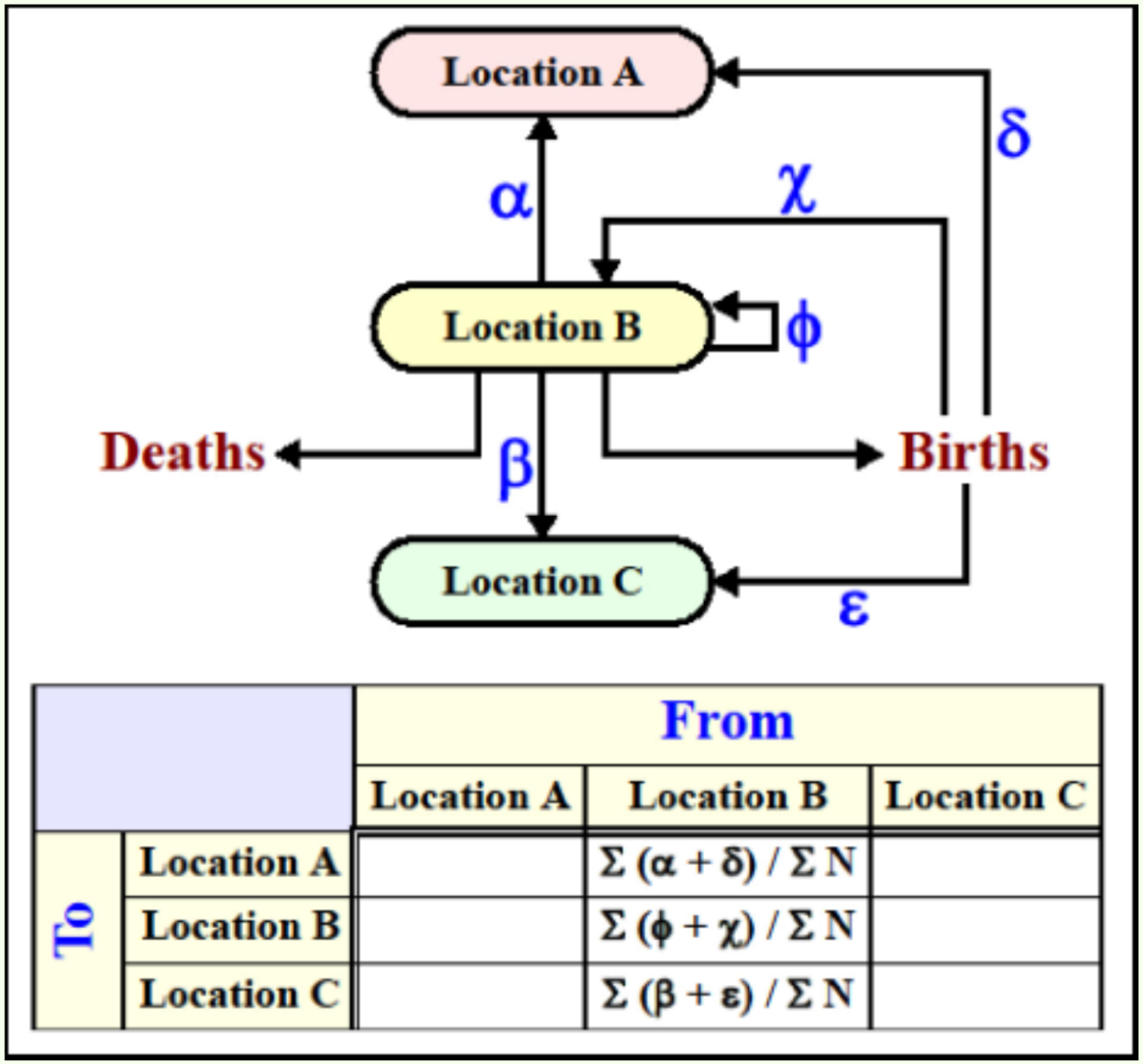
Productivity (10 Replicates)

Worst Sink — Neutral — Best Source

Strongest Source

# Measuring Landscape Connectivity

- Identify Locations of Interest
- Set Up HexSim to Record Locations
- Run Simulation
- Run a HexSim Location-Stratified Projection Matrix Report
- Assess Connectivity

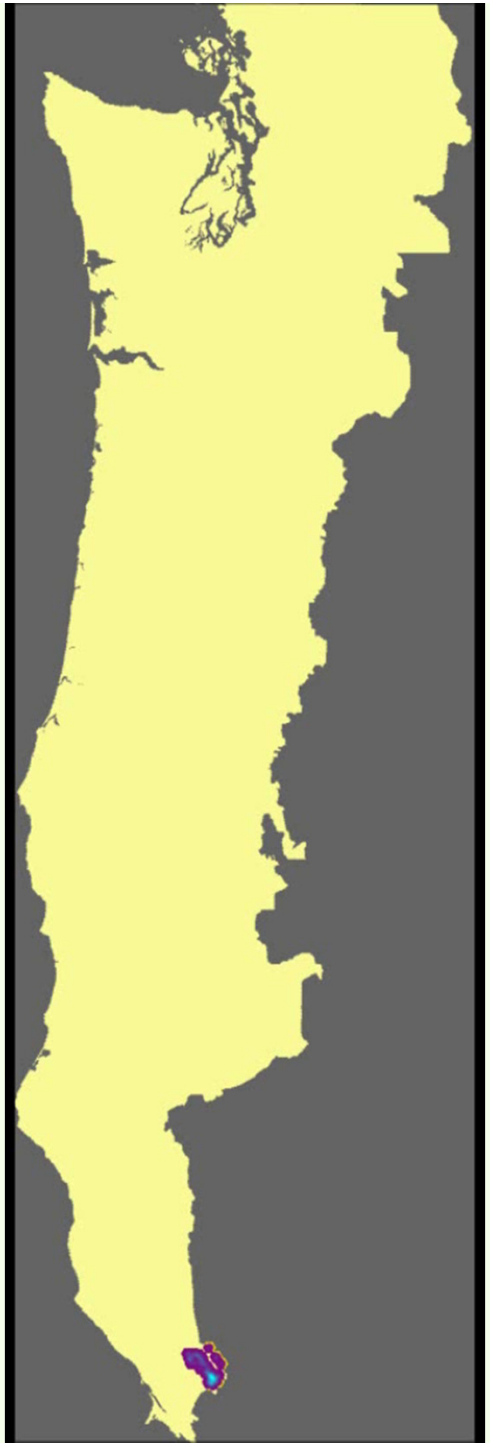



| | | From | | |
|---|---|---|---|---|
| | | Location A | Location B | Location C |
| To | Location A | | Σ (α + δ) / Σ N | |
| | Location B | | Σ (ϕ + χ) / Σ N | |
| | Location C | | Σ (β + ε) / Σ N | |

# National Forest Connectivity

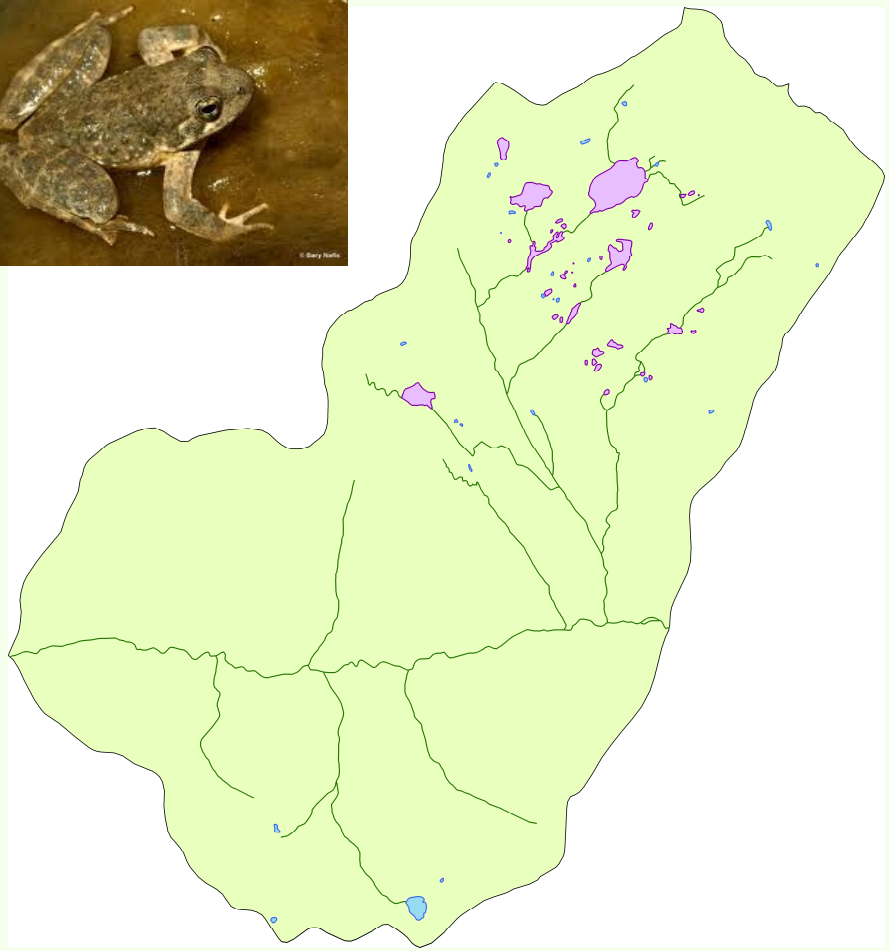
| | Mount Baker | Gifford Pinchot | Willamette | Rogue River | Shasta-Trinity | Olympic | Siskiyou | Okanogan | Suislaw | Deschutes | Winema | Six Rivers | Mendocino | Mount Hood | Wenatchee | Klamath | Umpqua |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Mount Baker | 0.9110 | 0.0001 | – | – | – | – | – | 0.0083 | – | – | – | – | – | – | 0.0153 | – | – |
| Gifford Pinchot | 0.0004 | 0.9461 | – | – | – | – | – | – | – | – | – | – | – | 0.0016 | 0.0056 | – | – |
| Willamette | – | – | 0.9501 | 0.0000 | – | – | – | – | 0.0001 | 0.0381 | – | – | – | 0.0190 | – | – | 0.0176 |
| Rogue River | – | – | 0.0000 | 0.8532 | – | – | 0.0087 | – | – | 0.0000 | 0.0560 | – | – | – | – | 0.0175 | 0.0199 |
| Shasta-Trinity | – | – | – | 0.0000 | 0.9157 | – | – | – | – | – | – | 0.0325 | 0.0130 | – | – | 0.0115 | – |
| Olympic | – | – | – | – | – | 0.7083 | – | – | – | – | – | – | – | – | – | – | – |
| Siskiyou | – | – | – | 0.0066 | – | – | 0.9122 | – | – | – | – | 0.0026 | – | – | – | 0.0046 | – |
| Okanogan | 0.0001 | – | – | – | – | – | – | 0.8738 | – | – | – | – | – | – | 0.0002 | – | – |
| Suislaw | – | – | 0.0000 | – | – | – | 0.0000 | – | 0.8692 | – | – | – | – | 0.0000 | – | – | – |
| Deschutes | – | – | 0.0052 | 0.0001 | – | – | – | – | – | 0.8989 | – | – | – | 0.0000 | – | – | 0.0005 |
| Winema | – | – | – | 0.0158 | – | – | – | – | – | 0.0000 | 0.8823 | – | – | – | – | 0.0000 | 0.0000 |
| Six Rivers | – | – | – | 0.0000 | 0.0234 | – | 0.0060 | – | – | – | – | 0.9000 | 0.0082 | – | – | 0.0276 | – |
| Mendocino | – | – | – | – | 0.0062 | – | – | – | – | – | – | 0.0042 | 0.9420 | – | – | – | – |
| Mount Hood | – | 0.0023 | 0.0059 | – | – | – | – | – | – | 0.0001 | – | – | – | 0.9487 | – | – | – |
| Wenatchee | 0.0399 | 0.0041 | – | – | – | – | – | 0.0094 | – | – | – | – | – | – | 0.9441 | – | – |
| Klamath | – | – | – | 0.0269 | 0.0063 | – | 0.0090 | – | – | – | 0.0001 | 0.0214 | – | – | – | 0.9011 | – |
| Umpqua | – | – | 0.0230 | 0.0309 | – | – | – | – | – | 0.0074 | 0.0001 | – | – | – | – | – | 0.9252 |

# Spotted Owl Connectivity Experiment



- Begin with an empty landscape
- Add 100 spotted owls in the south
- Run Simulation for 2500 years
- Record owl density
- Smooth density data and construct the video

# Frogs and the Chytrid Fungus



## Enzootic and epizootic dynamics of the chytrid fungal pathogen of amphibians

Cheryl J. Briggs[a,1], Roland A. Knapp[b], and Vance T. Vredenburg[c]

[a]Department of Ecology, Evolution and Marine Biology, University of California, Santa Barbara, CA 93106; [b]Sierra Nevada Aquatic Research Laboratory, University of California, Mammoth Lakes, CA 93546; and [c]Department of Biology, San Francisco State University, San Francisco, CA 94132

Edited* by David B. Wake, University of California, Berkeley, CA, and approved April 12, 2010 (received for review November 9, 2009)

**Chytridiomycosis, the disease caused by the chytrid fungus, *Batrachochytrium dendrobatidis* (Bd), has contributed to amphibian population declines and extinctions worldwide. The impact of this pathogen, however, varies markedly among amphibian species and populations. Following invasion into some areas of California's Sierra Nevada, Bd leads to rapid declines and local extinctions of frog populations (*Rana muscosa*, *R. sierrae*). In other areas, infected populations of the same frog species have declined but persisted at low host densities for many years. We present results of a 5-year study showing that infected adult frogs in persistent populations have low fungal loads, are surviving between years, and frequently lose and regain the infection. Here we put forward the hypothesis that fungal load dynamics can explain the different population-level outcomes of Bd observed in different areas of the Sierra Nevada and possibly throughout the world. We develop a model that incorporates the biological details of the Bd-host interaction. Importantly, model results suggest that host persistence versus extinction does not require differences in host susceptibility, pathogen virulence, or environmental conditions, and may be just epidemic and endemic population dynamics of the same host–pathogen system. The different disease outcomes seen in natural populations may result solely from density-dependent host–pathogen dynamics. The model also shows that persistence of Bd is enhanced by the long-lived tadpole stage that characterize these two frog species, and by nonhost Bd reservoirs.**

amphibian decline | *Batrachochytrium dendrobatidis* | chytridiomycosis | emerging infectious disease | host–pathogen dynamics

Chytridiomycosis, caused by the fungal pathogen *Batrachochytrium dendrobatidis* (Bd), has been called the "worst infectious disease ever recorded among vertebrates in terms of the number of species impacted, and its propensity to drive them to extinction" (1). Since it was first identified in the late 1990s (2, 3), Bd has been found in almost every region in which researchers have searched. It is now nearly global in its distribution, and it has been implicated in dramatic declines in amphibian populations worldwide (4, 5). One of the most striking features of this pathogen, however, is the variability in outcome of infection that has been observed among species, and among populations within a species. Chytridiomycosis leads to the rapid death of individuals of some species (2, 6, 7), whereas individuals of other species develop only minor infections and suffer little or no negative effects (8, 9). A number of factors, including temperature (10), innate defenses (11, 12), habitat (13, 14), and host life history traits (15), have been demonstrated to contribute to the variable outcomes of Bd infection.

Bd is currently having a devastating impact on populations of frogs in the mountain yellow-legged frog species complex (*Rana muscosa* and *R. sierrae*) in parts of the Sierra Nevada Mountains of California (6, 16). In Sequoia and Kings Canyon National Parks, we have documented the first appearance of Bd in many watersheds, resulting in the rapid decline of the frog populations (6, 16). The majority of these population crashes have caused the extirpation of all frog populations in the affected areas. However, a few populations, although reduced greatly in numbers after Bd arrival, were not extirpated (even those located adjacent to areas in which all frog populations have been extirpated). These mountain yellow-legged frog populations continue to be infected with Bd, but appear to be persisting with the fungus (17). In Yosemite National Park, the initial arrival of Bd was not observed; Bd has been present for at least a decade (18, 19). The remaining frog populations are all infected with Bd, but many appear to be persisting in the long term. Such vastly different dynamical outcomes of a pathogen (rapid extirpation vs. long-term persistence) suggest differences in the host–pathogen interaction at the different sites, for example, differences in frog susceptibility or fungal virulence. Here we propose that these types of differences might not be necessary to explain the observed varying outcomes of infection.

Mountain yellow-legged frogs occur only in high-elevation lakes and streams (above 1,500 m) in California. All stages of the frogs are aquatic, and in the Sierra Nevada, frogs spend 8–9 months of the year overwintering under ice. The tadpole stage is unusually long-lived, lasting 1–4 years. Although once abundant, these frogs have disappeared from most of their historic range during the past several decades (20). The spread of Bd is a major factor driving this decline (6, 16), with *R. muscosa* known to be infected with Bd since at least the 1970s (19).

Bd infects keratinized tissues of amphibians, specifically the skin of postmetamorphic stages and mouth parts of larval stages (3, 21). Bd is transmitted via an aquatic flagellated zoospore (21, 22). Zoospores are thought to infect cells within the stratum granulosum either directly or via a germ tube and then develop into sporangia (3, 21). After a temperature-dependent number of days, the sporangium releases zoospores through a discharge papilla (21, 23). Berger (21) showed through electron microscopy that discharge papillae usually point to the skin surface, suggesting that most zoospores are released to the outer surface of the skin, although some zoospores might stay within skin layers and potentially cause self-reinfection. Whereas other chytrid fungi have a sexual stage resulting in a thick-walled resistant sporangium, such a stage has not yet been identified in Bd (but see ref. 24). Bd usually has little detectable negative effect on infected tadpoles (25, 26), but Bd can lead to the death of postmetamorphic animals of many species within weeks of infection (2, 6, 27).

Here we investigate how infected *R. sierrae* populations are able to persist with Bd. We present a 5-year field study that reveals that adults in persistent populations are infected with only low-level infections (low Bd load), and individuals frequently lose and regain the infection. This is in stark contrast to

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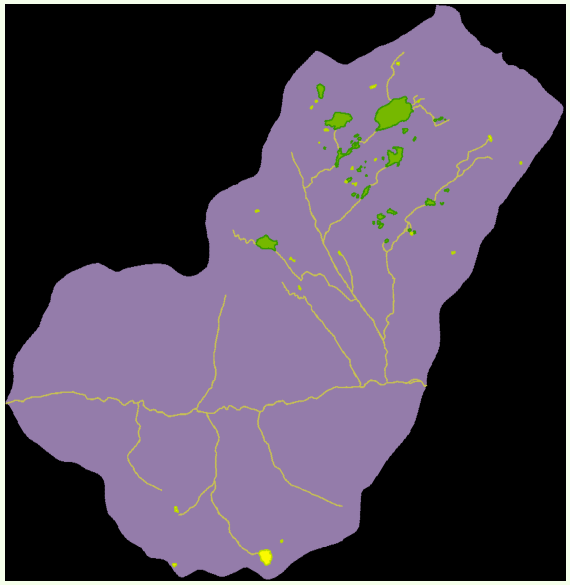
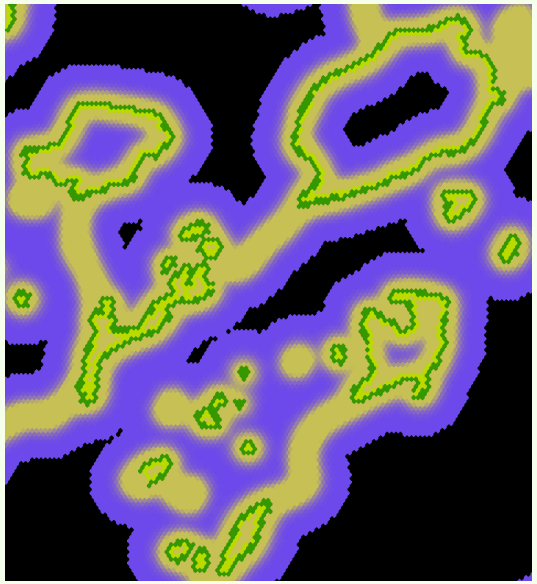
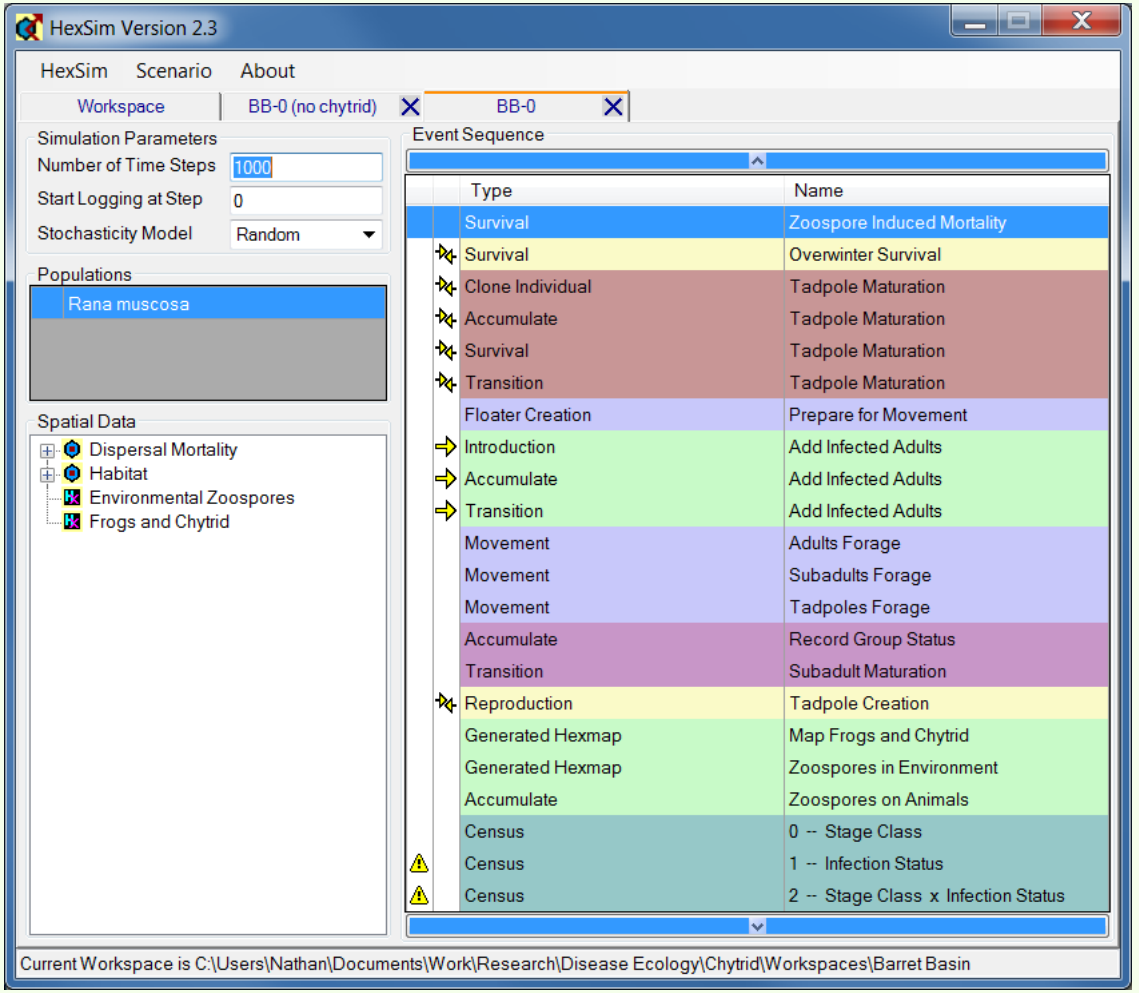
ECOLOGY

# HexSim Model of the Barrett Basin, CA Study Area

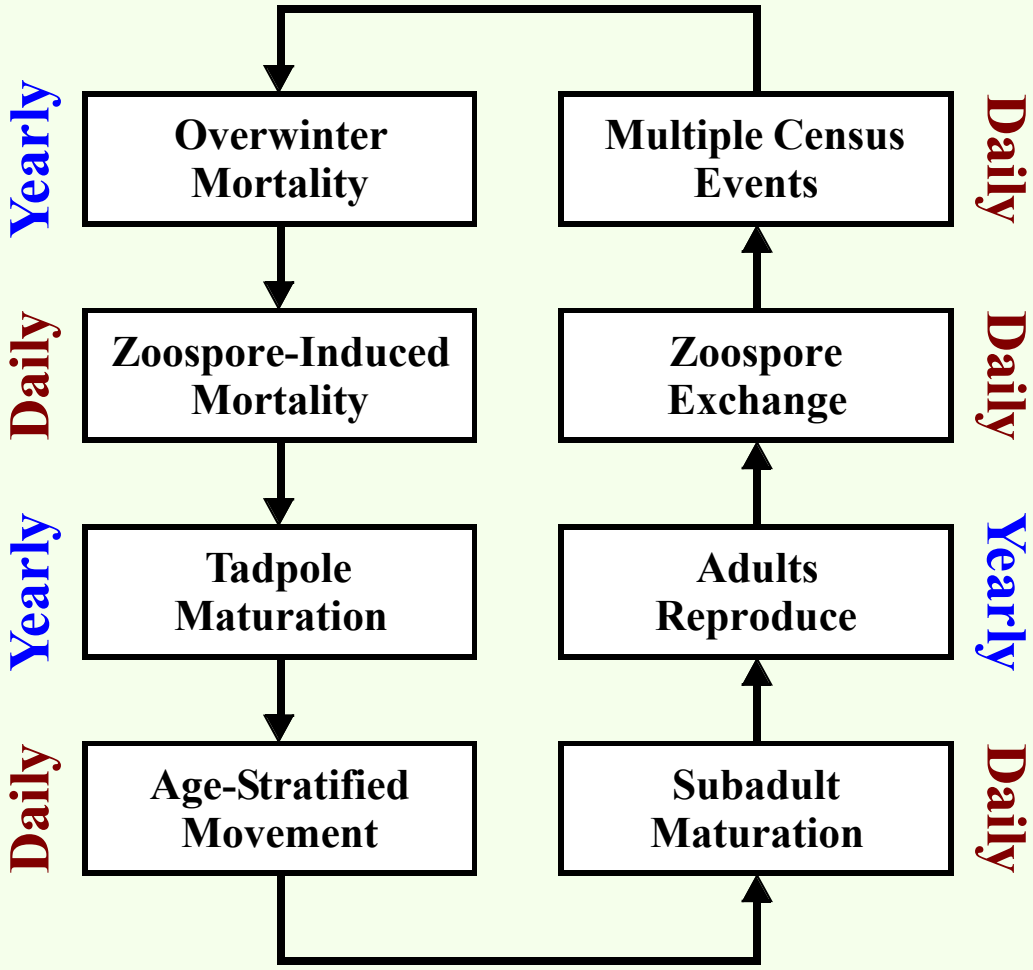




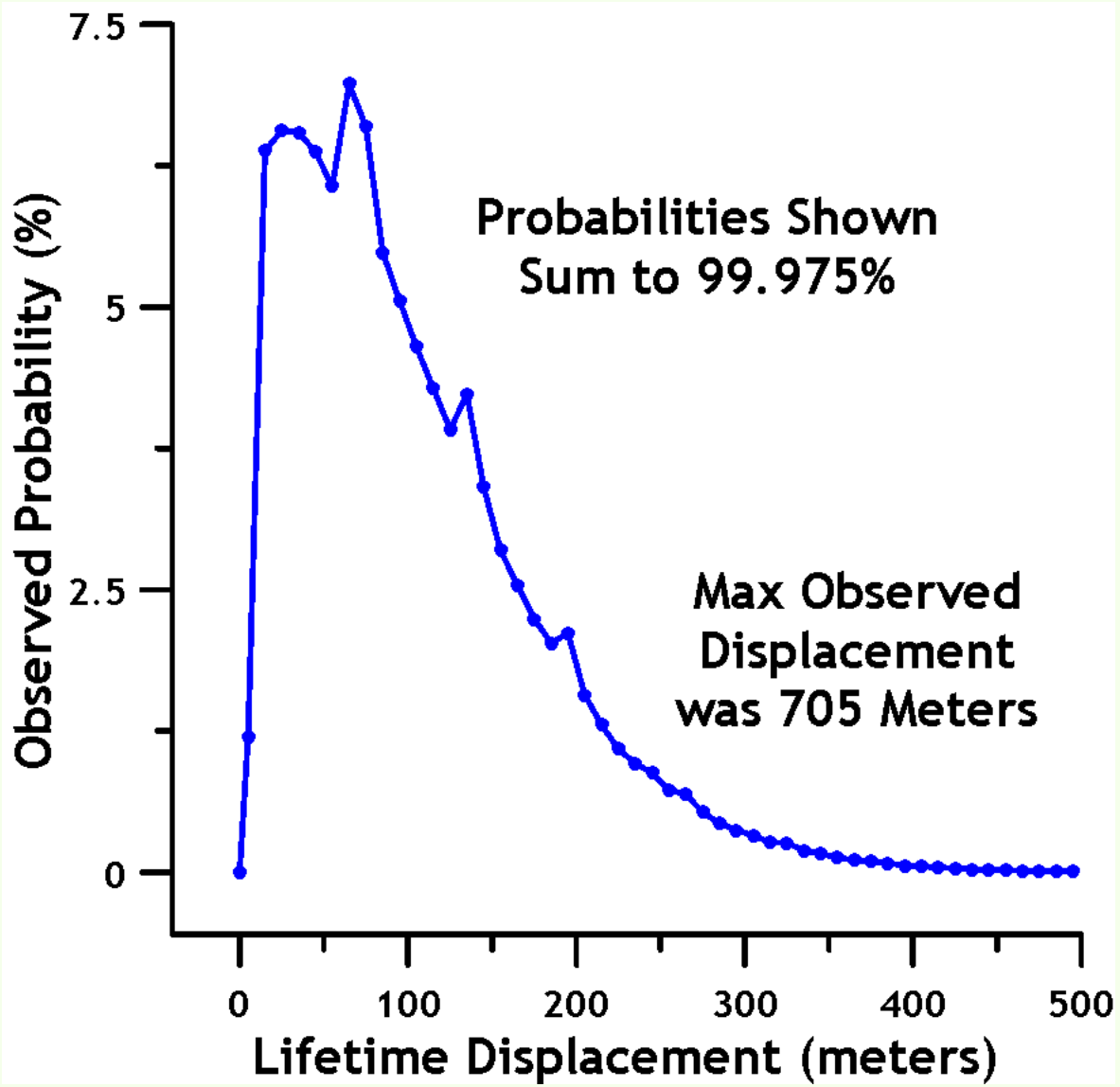


# Simulation Structure

- Adult mortality is 10% / year
- Tadpole & juvenile mortality is 30% / year
- To increase computation speed, we use “meta-tadpoles”
- Movement rules vary based on stage class
- Subadults need resources in order to mature
- Cytrid added at TS-60 Fungal exchange is to and from environment
- Fungus experiences mortality both on frogs and in the water



# Model Verification



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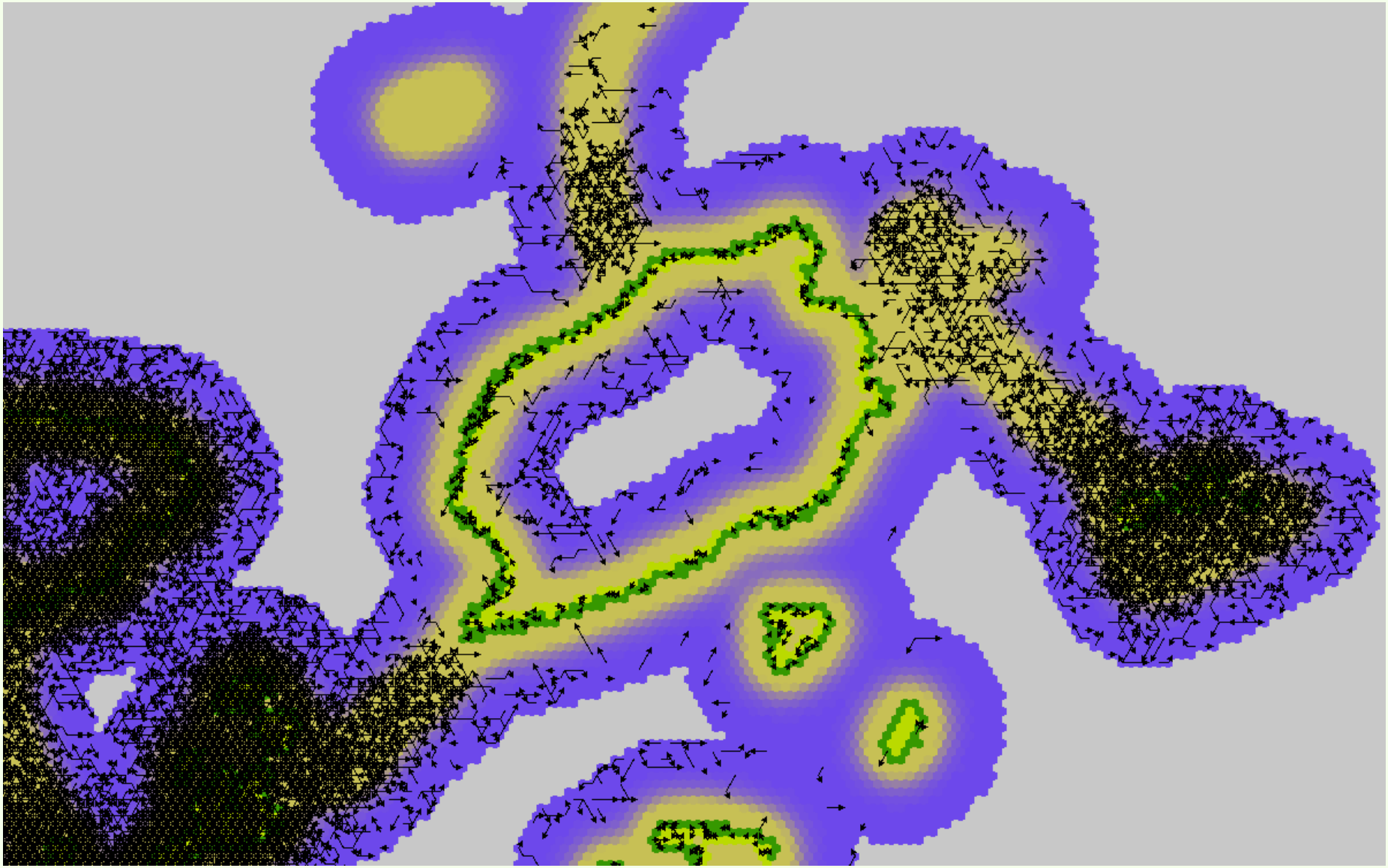
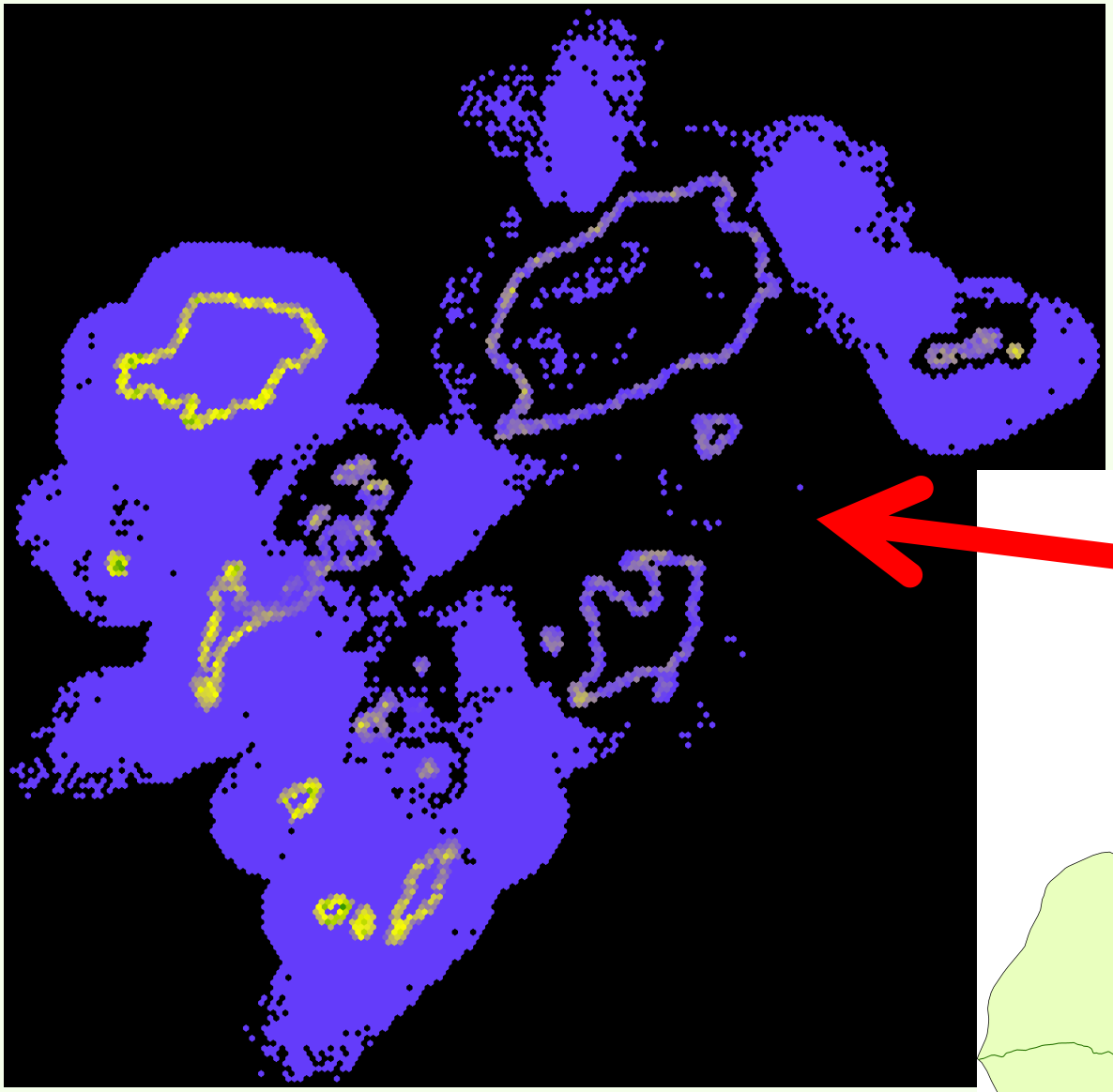
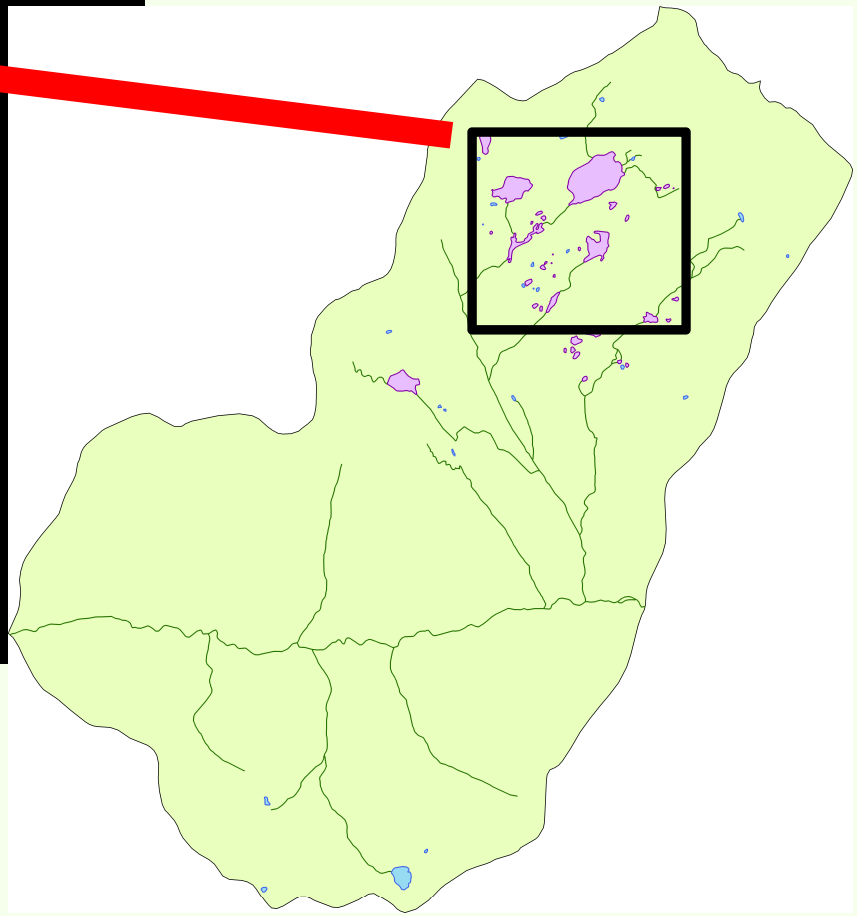
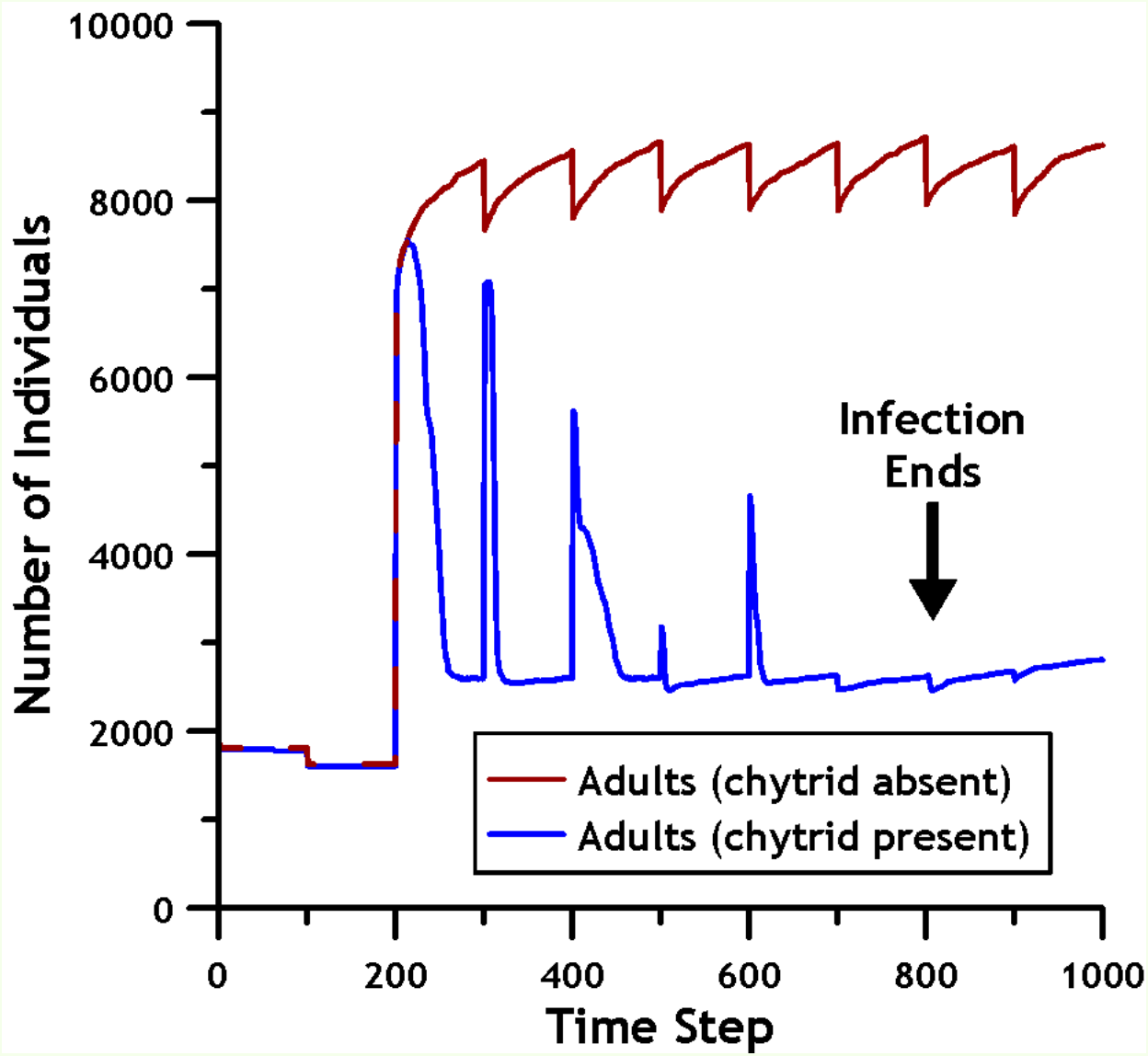


# Fungal Dynamics

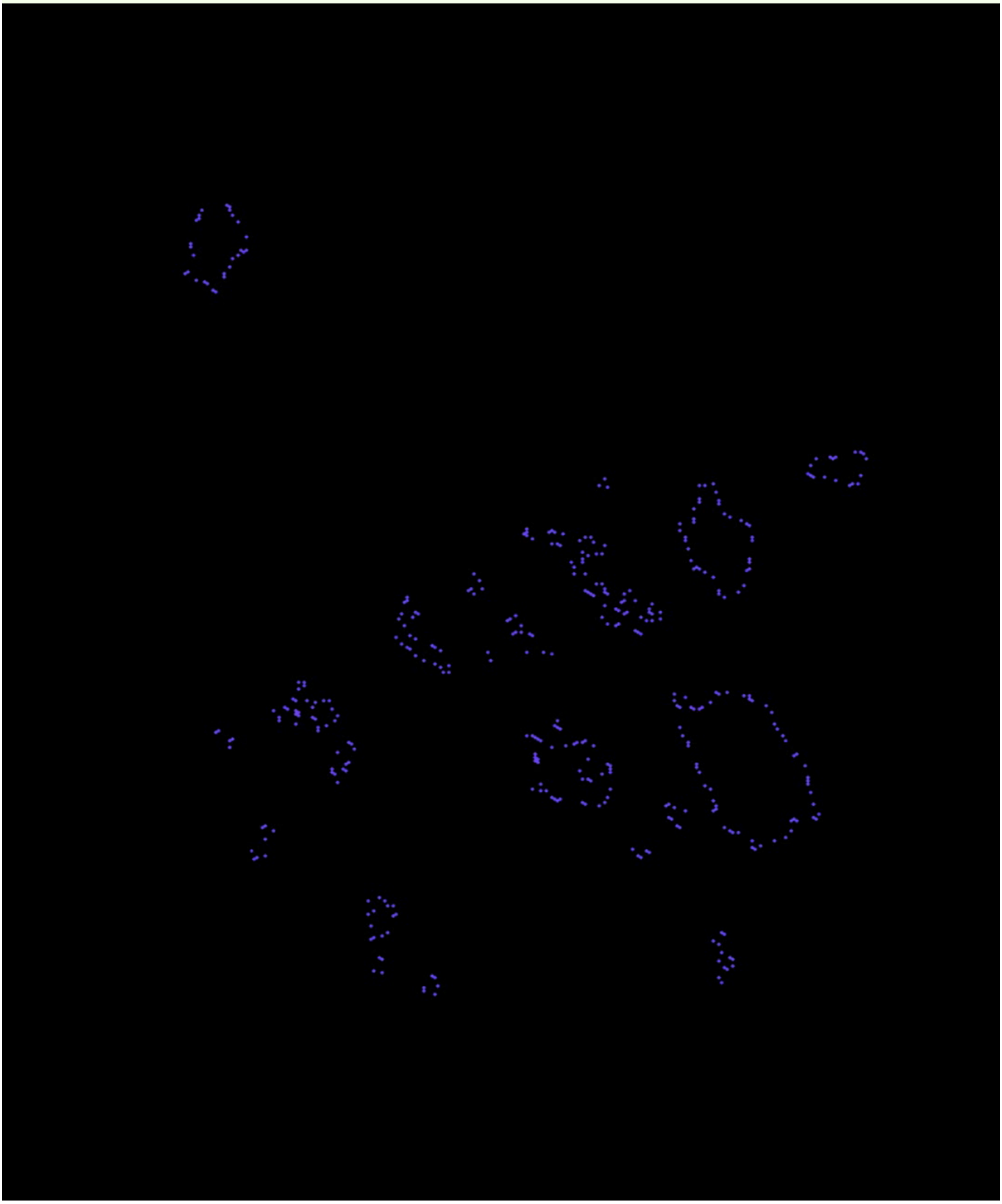




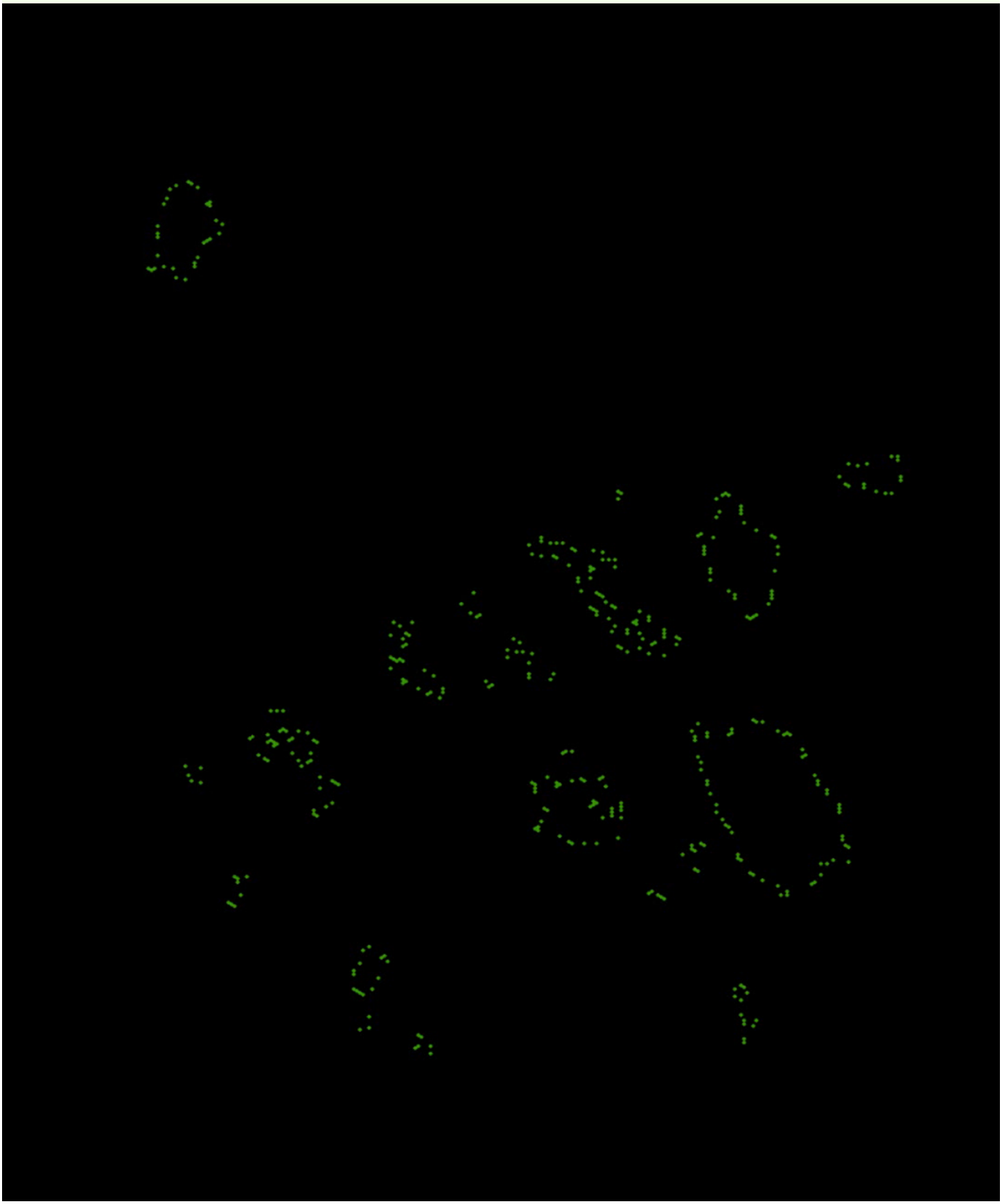
# Chytrid's Impact on Population Size



# Disease-Free Frog Dynamics



# Chytrid Invasion Dynamics



# Next Steps

## Model Comparison with Briggs et. Al. 2010…

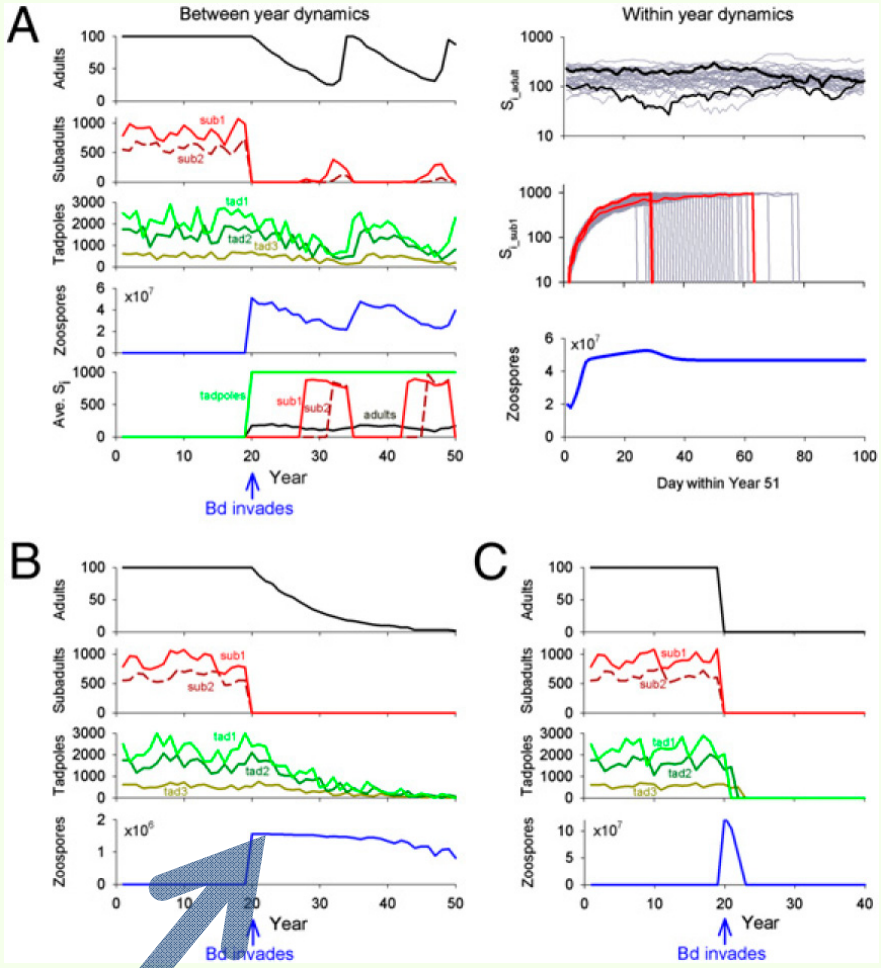
zoospores are calculated based on the numbers at the start of the time step as follows. At a given number of zoospores, $Z$, $dS_i/dt = (\beta/V)\ Z + c_1\ S_i(t)$, where $c_1 = \eta\nu\ f - \sigma$, such that

$$S_i(t+\Delta t) = \frac{c_1 e^{c_1 \Delta t} S_i(t) + (\frac{\beta}{V})(e^{c_1 \Delta t} - 1)Z}{c_1}. \qquad \textbf{[S1]}$$

At a given distribution of sporangia, $S_i$, across the $N$ frogs currently alive in the population, $dZ/dt = c_2 - c_3\ Z(t)$, where $c_2 = \sum_{i=1}^{N}[\eta(1-f)S_i]$ and $c3 = (\gamma/V)\ N + \mu$, such that

$$Z(t+\Delta t) = \frac{e^{-c_3 \Delta t}\{c_2(e^{c_3 \Delta t} - 1) + c_3 Z(t)\}}{c_3} \qquad \textbf{[S2]}$$

For small numbers of sporangia on a frog, $S_i(t + \Delta t)$ was replaced with $\text{Pois}\{S_i(t+\Delta t)\}$, a draw from a Poisson distribution with mean $= S_i(t + \Delta t)$, to incorporate the effects of demographic




## Briggs et. Al. model

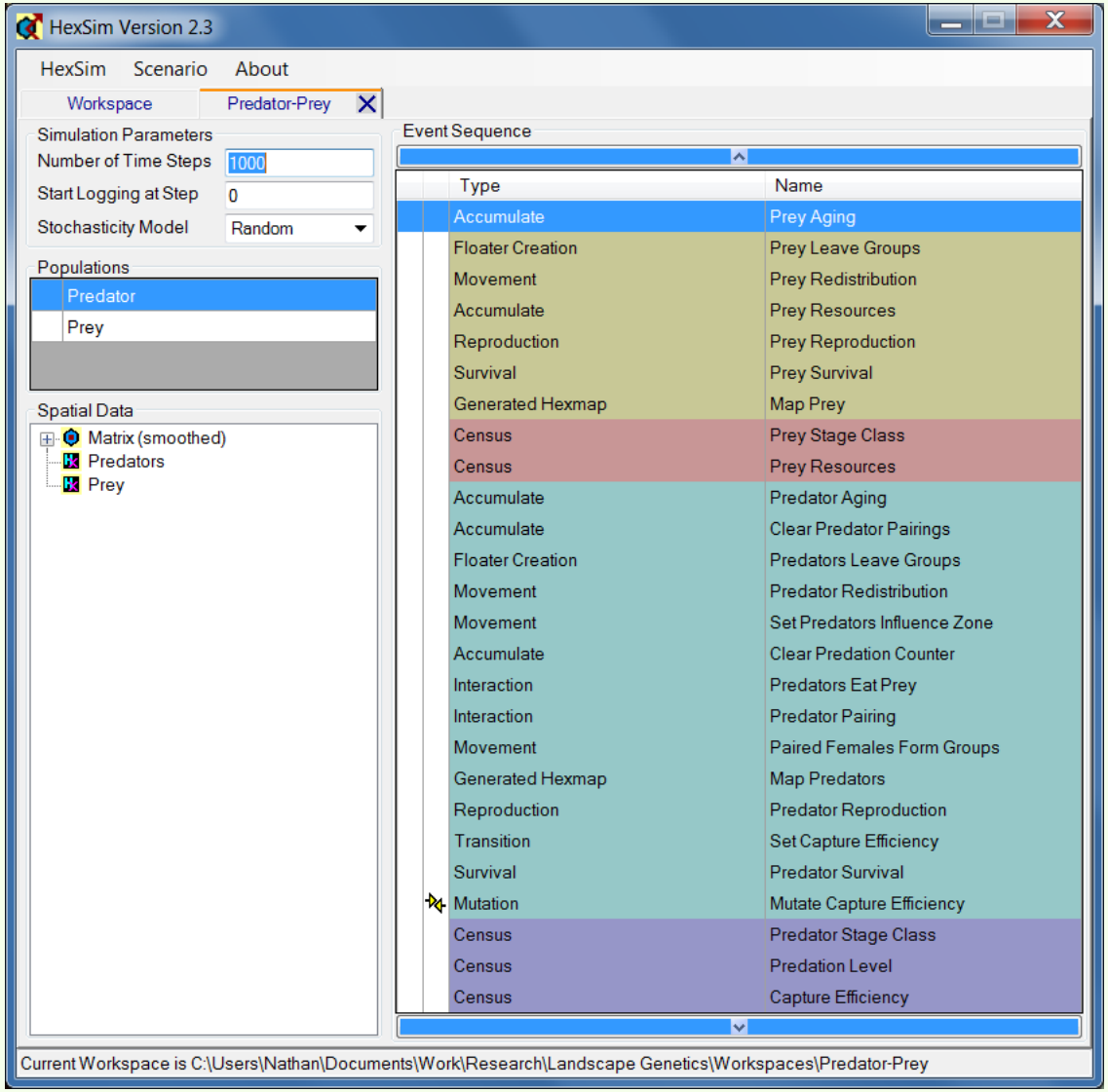
# Landscape Genetics

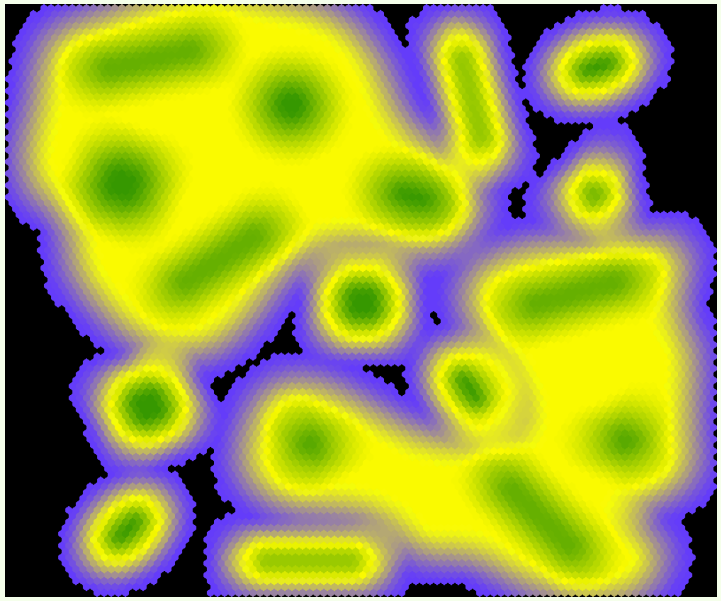
- Each individual is assigned a genome
- Populations can have any number of loci
- Each locus can have any number of alleles
- Users define initial allele spatial distributions
- Map distance may be used to simulate chromosome crossover
- Traits can be neutral or adaptive
- Mutation events modify individual genotype

# Example: Predators & Prey

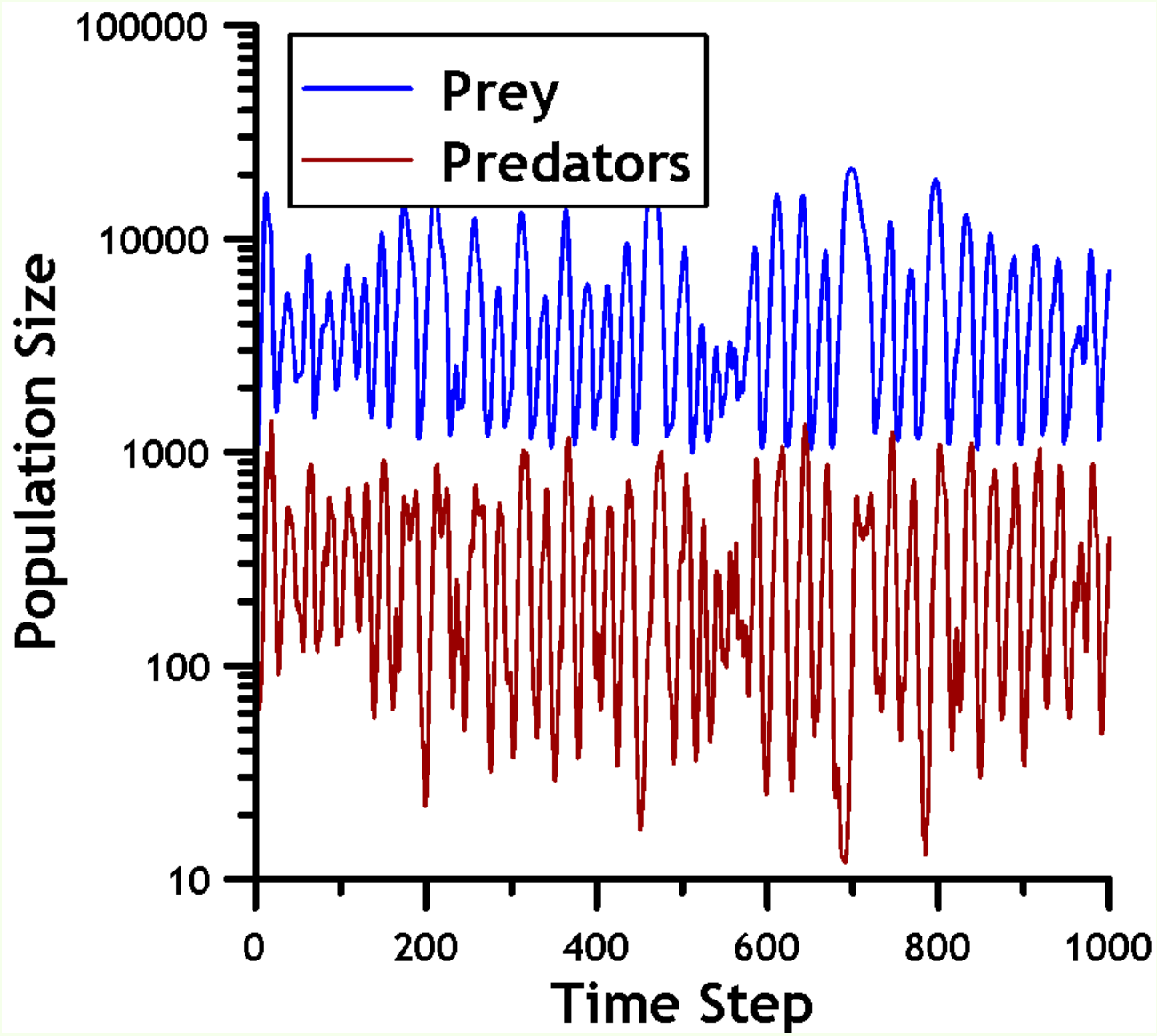
- Two interacting populations
- Predators and prey use different mating schemes
- Prey live in colonies, predators are solitary
- Predators disperse towards prey
- Predator capture efficiency is controlled through a heritable trait
- Capture efficiency influences both survival and reproduction
- Semi-random mutations alter the capture efficiency gene

# HexSim Predator-Prey Model

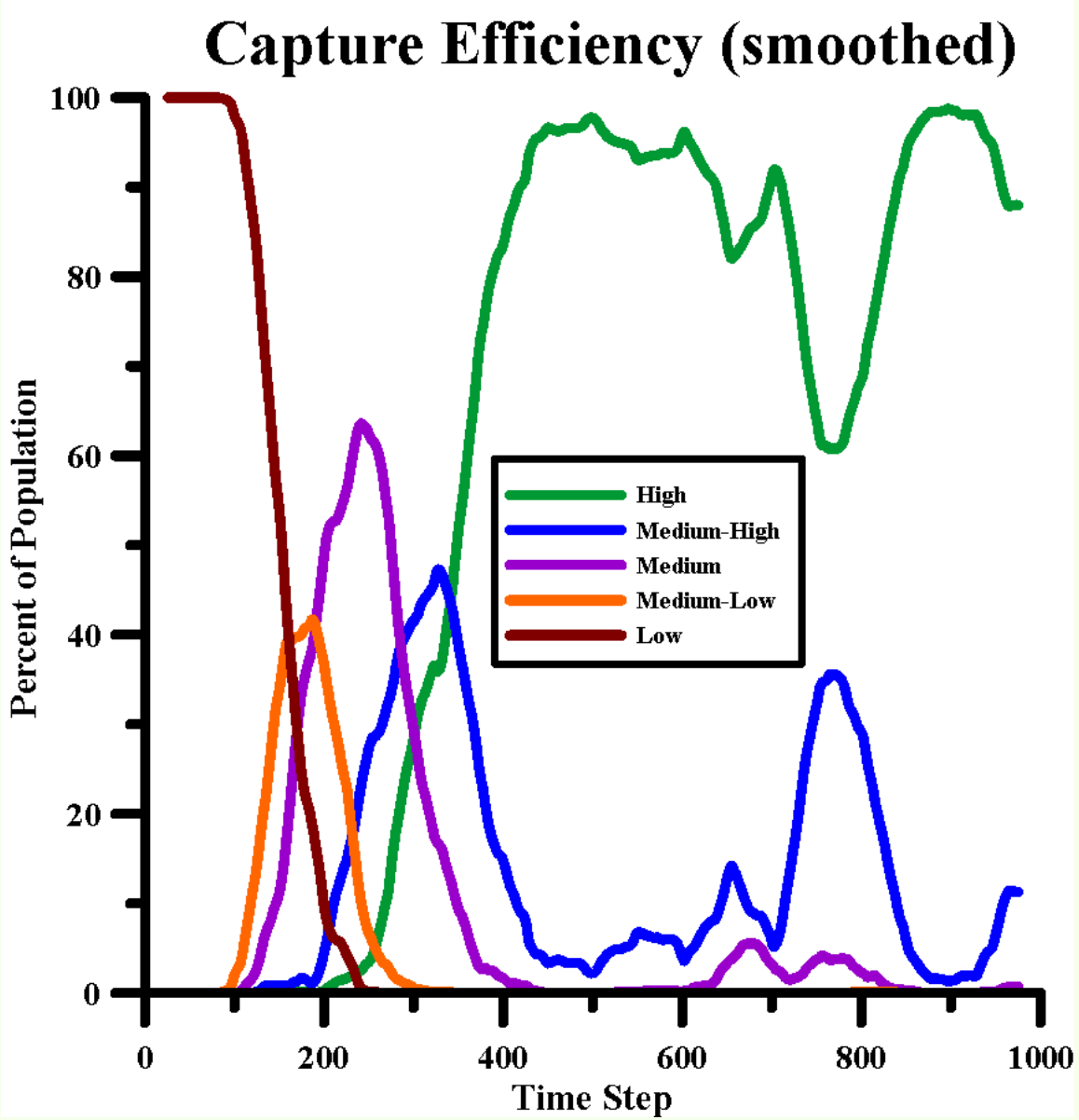


# Population Fluctuations

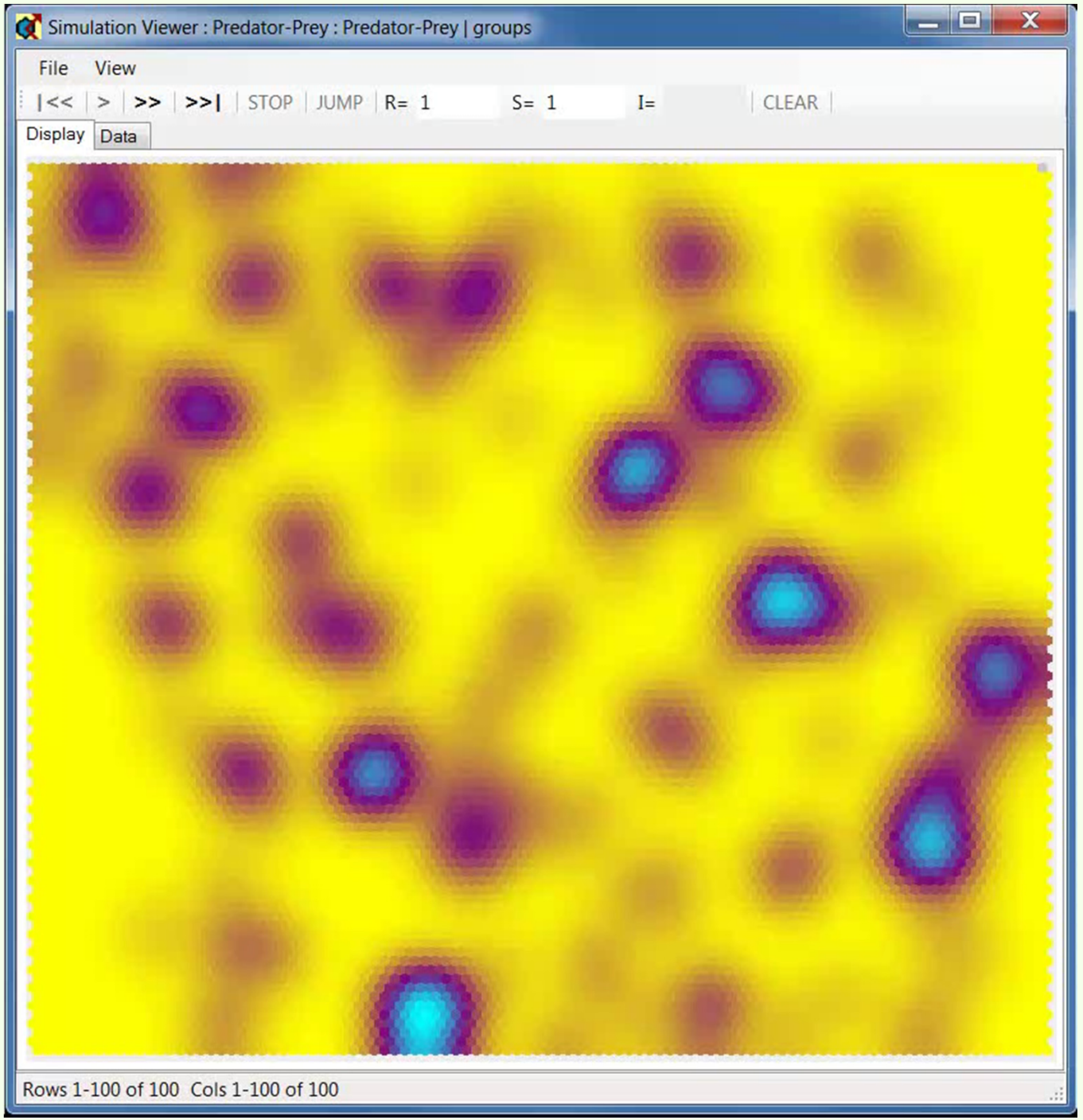


# Selective Pressure Ensures High Capture Efficiency



# Predator Control of the Prey Population

**Note presence of two prey refuges**



# In Summary

**The use of sophisticated simulation models in Conservation Biology and other disciplines is going to increase with time…**

**In particular, new spatially-explicit, individual-based models are creating opportunities to improve conservation theory.**

**Its important that users of these tools make an effort to:**

- **Use the simplest models that are capable of capturing critical processes**
- **Ensure that the models they construct are defensible**
- **Conduct model comparisons**
- **Contribute to the model development process (the model builders need your expertise)**